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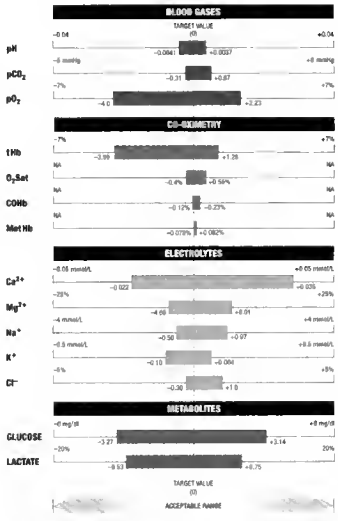

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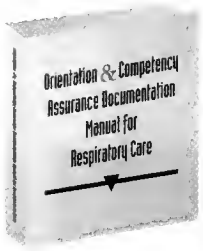

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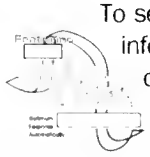

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Newer Asthma Therapies (editorial)—Smith LJ. Ann Intern Med 1999;130(6):531–532.

Prevalence of Acute Respiratory Distress Syndrome after Cardiac Surgery—Asimakopoulos G, Taylor KM, Smith PL, Ratnatunga CP. J Thorac Cardiovasc Surg 1999;117(3):620–621.

Is Informed Consent Always Necessary for Randomized, Controlled Trials?—Truog RD, Robinson W, Randolph A, Morris A. N Engl J Med 1999;340(10):804–807.

Economic Implications of the Diagnosis of Obstructive Sleep Apnea (editorial)—Pack AI, Gurubhagavatula I. Ann Intern Med 1999;130(6):533–534.

Practice Guidelines for Preoperative Fasting and the Use of Pharmacologic Agents to Reduce the Risk of Pulmonary Aspiration: Application to Healthy Patients Undergoing Elective Procedures. A report by the American Society of Anesthesiologist Task Force on Preoperative Fasting–Anesthesiology 1999;90(3):896–905.

Preventing Complications during Percutaneous Tracheostomy—Bouvette M, Fuhrman TM. Anesthesiology 1999;90(3):918–919.

The Cuffed Oropharyngeal Airway and Management of the Difficult Airway—Patel A, Pearce A. Anesthesiology 1999;90(3):924–925.

The WuScope Technique for Endotracheal Tube Exchange—Andrews SR, Norcross SD, Mabey MF, Siegel JB. Anesthesiology 1999;90(3):929–930.

Infection Control in Long-Term Care: News from the Front—Strausbaugh LJ. Am J Infect Control 1999;27(1):1–3.

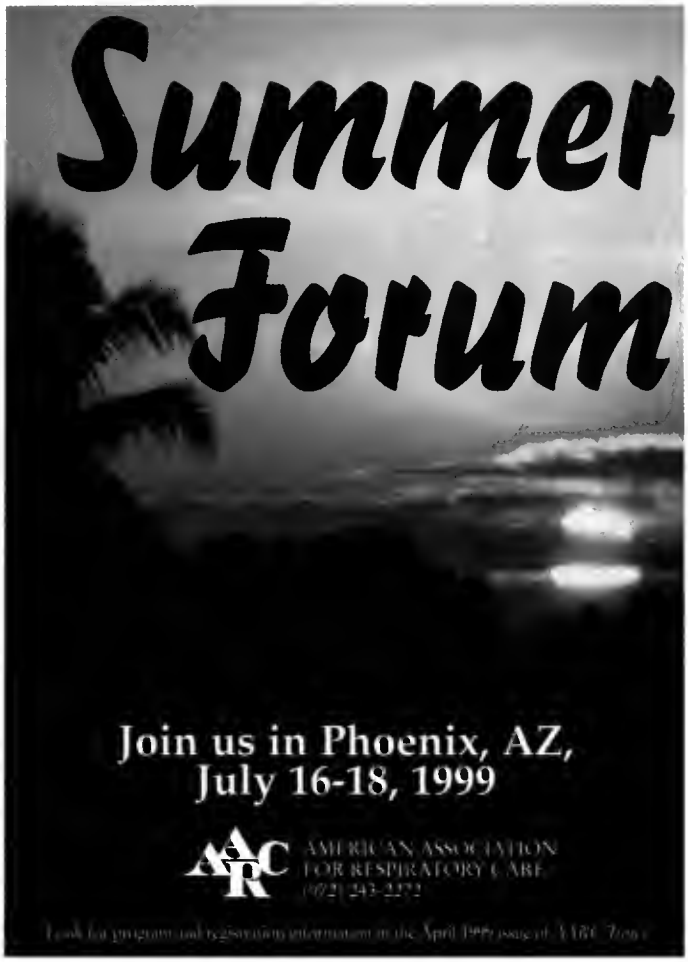
Team Approach to Infection Prevention and Control in the Nursing Home Setting—Ahlbrecht H, Shearen C, Degelau J, Guay DRP. Am J Infect Control 1999;27(1):64–70.

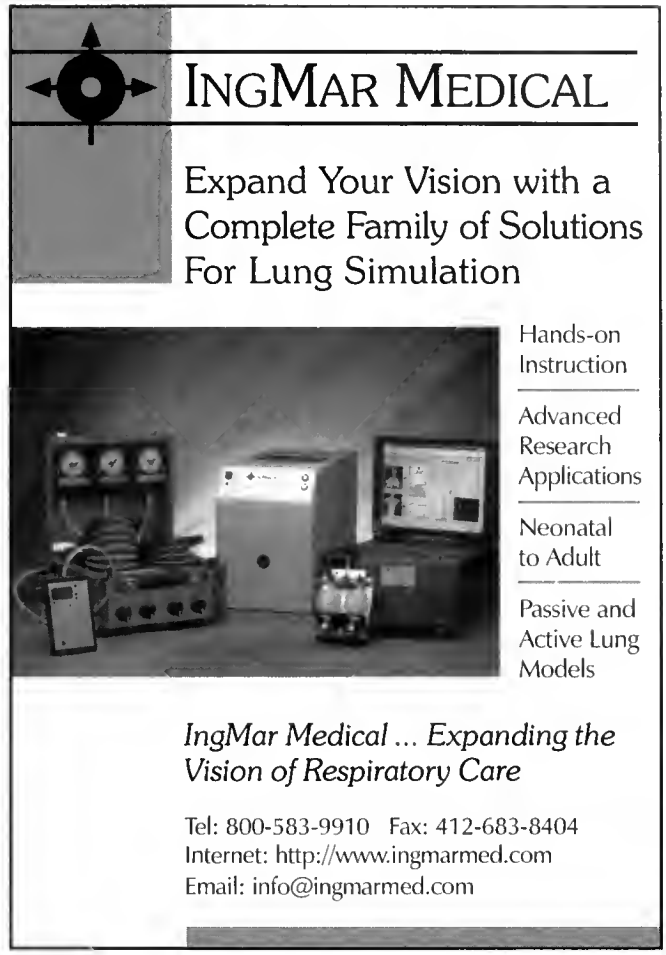
Large Scale Implementation of a Respiratory Therapist-Driven Protocol for Ventilator Weaning—Ely EW, Bennett PA, Bowton DL, Murphy SM, Florance AM, Haponik EF. Am J Respir Crit Care Med 1999;159(2):439.

We prospectively investigated the large-scale implementation of a respiratory-therapist-driven protocol (TDP) that included 117 respiratory care practitioners (RCPs) managing 1,067 patients with respiratory failure over 9,048 patient days of mechanical ventilation. During a 12-mo period, we reintroduced a previously validated protocol that included a daily screen (DS) coupled with spontaneous breathing trials (SBTs) and physician prompt, as a TDP without daily input from a physician or "weaning team." With graded, staged educational interventions at 2-mo intervals, RCPs had a 97% completion rate and a 95% correct interpretation rate for the DS. The frequency with which patients who passed the DS underwent SBTs increased throughout the implementation process ($p < 0.001$). As the year progressed, RCPs more often considered SBTs once patients had passed a DS ($p < 0.001$), and physicians ordered more SBTs (46 versus 65%, $p = 0.004$). Overall, SBTs were ordered more often on the medicine than on the surgical services (81 versus 63%, $p = 0.001$), likely reflecting medical intensivists' prior use of this protocol. Important barriers to protocol compliance were identified through a questionnaire (89 respondents, 76%), and included: Physician unfamiliarity with the protocol, RCP inconsistency in seeking an order for an SBT from the physician, specific reasons cited by the physician for not advancing the patient to a SBT, and lack of stationary unit assignments by RCPs performing the protocol. We conclude that implementation of a validated weaning strategy is feasible as a TDP without daily supervision from a weaning physician or team. RCPs can appropriately perform and interpret DS data more than 95% of the time, but significant barriers to SBTs exist. Through a staged implementation process, using periodic reinforcement of all participants in ventilator management, improved compliance with this large-scale weaning protocol can be achieved.

Nicotine Nasal Spray with Nicotine Patch for Smoking Cessation: Randomised Trial with Six Year Follow Up—Blondal T, Gudmundsson LJ, Olafsdottir I, Gustavsson G, Westin A. BMJ 1999;318(7179):285.

Objective: To evaluate the efficacy of using a nicotine patch for 5 months with a nicotine nasal spray for 1 year. Design: Placebo controlled, double blind trial. Setting: Reykjavik health centre. Subjects: 237 smokers aged 22–66 years living in or around Reykjavik. Interventions: Nicotine patch for 5 months with nicotine nasal





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spray for 1 year (n=118) or nicotine patch with placebo spray (n=119). Treatment with patches included 15 mg of nicotine for 3 months, 10 mg for the fourth month, and 5 mg for the fifth month, whereas nicotine in the nasal spray was available for up to 1 year. Both groups received supportive treatment. Main outcome measure: Sustained abstinence from smoking. Results: The log rank test for 6 years (X^2=8.5, P=0.004) shows a significant association between abstinence from smoking and type of treatment. Sustained abstinence rates for the patch and nasal spray group and patch only group were 51% v 35% after 6 weeks (P=0.011 (X^2), 95% confidence interval 1. 17% to 3.32%), 37% v 25% after 3 months (P=0.045, 1.01% to 3.08%), 31% v 16% after 6 months (P=0.005, 1.27% to 4.50%), 27% v 11% after 12 months (P=0.001, 1.50% to 6.14%), and 16% v 9% after 6 years (P=0.077, 0.93% to 4.72%). Conclusions: Short and long term abstinence rates show that the combination of using a nicotine patch for 5 months with a nicotine nasal spray for 1 year is a more effective method of stopping smoking than using a patch only. The low percentage of participants using the nasal spray at 1 year, and the few relapses during the second year, suggest that it is not cost effective to use a nasal spray for longer than 7 months after stopping a patch.

Endobronchial Intubation Causes an Immediate Increase in Peak Inflation Pressure in Pediatric Patients—Campos C, Naguib SS, Chuang AZ, Lemak NA, Khalil SN. Anesth Analg 1999;88(2):268.

Our purpose was to determine whether endobronchial intubation always causes an immediate increase in peak inflation pressure and, if so, the magnitude of the increase. Fourteen children scheduled for central line placement for prolonged antibiotic administration comprised the study group. After routine premedication and induction of anesthesia (halothane in oxygen), an endotracheal tube was inserted, and its position was verified by auscultation and fluoroscopy. Children were mechanically ventilated using a preset volume pressure-limited ventilator with a 5-L fresh gas flow. All children received a constant tidal volume using a similar circuit, similar tubing, and a similar compression volume. The lowest peak inflation pressure to deliver a tidal volume of 15 mL/kg was used. After adjusting the respiratory rate (end-tidal CO_2 30 mm Hg) and anesthetic level (halothane end-tidal 1.2%), the peak inflation pressure at this endotracheal position was recorded. The endotracheal tube was advanced into a bronchus, the position was verified as above, and peak inflation pressure was recorded. The endobronchial tube was then pulled back into the trachea, and placement of the central line proceeded. The peak inflation pressure at the endobronchial position was significantly greater than the peak inflation pressure at the endotracheal position ($P < 0.0001$). The increase was instantaneous at the endobronchial position. Monitoring peak inflation pressure while inserting an endotracheal tube and during anesthesia can help to diagnose endobronchial intubation. Implications: Monitoring peak inflation pressure while inserting an endotracheal tube and during anesthesia can help to diagnose endobronchial intubation.

How Does Home Management of Asthma Exacerbations by Parents of Inner-City Children Differ from NHLBI Guideline Recommendations?—Warman KL, Silver EJ, McCourt MP, Stein REK. Pediatrics 1999; 103(2):422.

Objectives. 1) To describe the asthma morbidity, primary care practices, and asthma home management of inner-city children with asthma; 2) to determine the responses of parental caretakers to asthma exacerbations in their child; and 3) to compare these responses to the recommendations of the National Heart, Lung, and Blood Institute (NHLBI) asthma guidelines for home management of acute exacerbations of asthma. Design and Methods. A 64-item telephone survey was administered between July 1996 and June 1997 to 220 parental caretakers of 2- to 12-year-old children who had been hospitalized with asthma at an inner-city medical

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T O R S

center from January, 1995 to February, 1996. Sociodemographics, primary care practices, asthma morbidity, and asthma home management were assessed. Parents were asked what they would do if their child "began wheezing and breathing faster than usual." Results. Morbidity measures indicated that there were an average of 2.5 ± 4.5 emergency department visits for asthma in the last 6 months, 1.6 ± 2.2 hospitalizations for asthma in the last 12 months, and 18.1 ± 17.9 asthma-related school absences in the previous school year. Most, but not all, of the families had primary care providers and most had phone access to them. Half of the families (51%) reported having been given a written asthma action plan. Only 30% of families with children age 5 years and older had peak flow meters. In contrast, almost all families (97%) had equipment for inhalation of β-agonists. Only 39% of the 181 children with persistent symptoms were receiving daily antiinflammatory agents as recommended in the guidelines of the NHLBI. In response to the scenario of an acute exacerbation of asthma, no one mentioned that they would refer to a written plan, only 1 caretaker would measure peak flow and 36% would give β-agonists. Two percent would give oral steroids initially, and 1 additional person would do so if wheezing continued 40 minutes later. Only 4% responded that they would contact their clinician. Reports of actual practice differed from the scenario responses in that more people began β-agonists and oral steroids in response to an exacerbation in the past 6 months than said they would in response to the scenario. Conclusion. In this population of previously hospitalized inner-city children with asthma, the NHLBI guidelines for the home management of asthma exacerbations are not being followed. Interventions are needed to affect both clinician and caretaker practices.

Clinical Use of Continuous Arterial Blood Gas Monitoring in the Pediatric Intensive Care Unit—Weiss IK, Fink S, Harrison R, Feldman JD, Brill JE. Pediatrics 1999;103(2):440.

Context. Continuous arterial blood gas monitoring is a new technology based on the combination of opto-chemical and fiber-optic detectors that can measure pH, PCO_2, PO_2, and temperature on a continuous basis via a sensor placed in an artery. Objective. To evaluate this technology in pediatric patients who would normally require frequent arterial blood gas sampling. Design. A criterion standard study in which the results of arterial blood gas samples measured by the laboratory analyzer were compared with the sensor readings. Setting. A pediatric intensive care unit of a tertiary referral center. Patients. Children with severe respiratory failure who required frequent arterial blood gas sampling and who had a 20-gauge arterial line in either a radial or femoral site. Results. Twenty-four patients with a mean age of 6.4 years (range 1–21 years) had a sensor placed. Sensors were in place for a mean of 101 ± 62 hours. Eighteen patients underwent continuous monitoring for at least 24 hours or until no longer clinically necessary. There were 414 pairs of blood gas samples obtained. The bias/precision for pH was 0.005/0.030; for PCO_2, -1.8/6.3 mm Hg; and for PO_2, 1.2/24 mm Hg. The correlation (r value) between the sensor readings and the blood gases were pH 0. 960, PCO_2 0.927, PO_2 0.813 ($P < 0.01$ for all values). The bias and precision for PO_2 levels < 70 mm Hg were 0.057/9.34 mm Hg. There were no complications from sensor placement. Continuous blood gas monitoring allowed immediate recognition of clinical changes. Conclusion. The continuous arterial blood gas sensor is capable of clinically accurate blood gas measurements. This technology provides the clinician with immediate data that can allow rapid interventions in unstable patients.

Noninvasive Ventilation for the Treatment of Acute Respiratory Failure in Patients with Hematologic Malignancies: A Pilot Study—Conti G, Marino P, Cogliati A, Dell'Utri D, Lappa A, Rosa G, Gasparetto A. Intensive Care Med 1998;24(12):1283.

OBJECTIVE: To evaluate treatment with noninvasive ventilation (NIV) by nasal mask as an alternative to endotracheal intubation and conventional mechanical ventilation in patients with hematologic malignancies complicated by acute respiratory failure to decrease the risk of hemorrhagic complications and increase clinical tolerance. DESIGN: Prospective clinical study. SETTING: Hematologic and general intensive care unit (ICU), University of Rome "La Sapienza". PATIENTS: 16 consecutive patients with acute respiratory failure complicating hematologic malignancies. INTERVENTIONS: NIV was delivered via nasal mask by means of a BiPAP ventilator (Respironics, USA); we evaluated the effects on blood gases, respiratory rate, and hemodynamics along with tolerance, complications, and outcome. MEASUREMENTS AND RESULTS: 15 of the 16 patients showed a significant improvement in blood gases and respiratory rate within the first 24 h of treatment. Arterial oxygen tension (P_{aO_2}), P_{aO_2}/F_{IO_2} (fractional inspired oxygen) ratio, and arterial oxygen saturation significantly improved after 1 h of treatment (43±10 vs 88±37 mmHg; 87±22 vs 175±64; 81±9 vs 95±4%, respectively) and continued to improve in the following 24 h ($p < 0.01$). Five patients died in the ICU following complications independent of the respiratory failure, while 11 were discharged from the ICU in stable condition after a mean stay of 4.3±2.4 days and were discharged in good condition from the hospital. CONCLUSIONS: NIV by nasal mask proved to be feasible and appropriate for the treatment of respiratory failure in hematologic patients who were at high risk of intubation-related complications.

A Controlled Trial of Exercise Rehabilitation after Heart Transplantation—Kobashigawa JA, Leaf DA, Lee N, Gleeson MP, Liu H, Hamilton MA, et al. N Engl J Med 1999;340(4):272.

BACKGROUND: In patients who have received a cardiac transplant, the denervated donor heart responds abnormally to exercise and exercise tolerance is reduced. The role of physical exercise in the treatment of patients who have undergone cardiac transplantation has not been determined. We assessed the effects of training on the capacity for exercise early after cardiac transplantation. METHODS: Twenty-seven patients who were discharged within two weeks after receiving a heart transplant were randomly assigned to participate in a six-month structured cardiac-rehabilitation program (exercise group, 14 patients) or to undergo unstructured therapy at home (control group, 13 patients). Each patient in the exercise group underwent an individualized program of muscular-strength and aerobic training under the guidance of a physical therapist, whereas control patients received no formal exercise training. Cardiopulmonary stress testing was performed at base line (within one month after heart transplantation) and six months later. RESULTS: As compared with the control group, the exercise group had significantly greater increases in peak oxygen consumption (mean increase, 4.4 mL per kilogram of body weight per minute [49 percent] vs. 1.9 mL per kilogram per minute [18 percent]; P=0.01) and workload (mean increase, 35 W [59 percent] vs. 12 W [18 percent]; P=0.01) and a greater reduction in the ventilatory equivalent for carbon dioxide (mean decrease, 13 [20 percent] vs. 6 [11 percent]; P=0.02). The mean dose of prednisone, the number of patients taking antihypertensive medications, the average number of episodes of rejection and of infection during the study period, and weight gain did not differ significantly between the groups. CONCLUSIONS: When initiated early after cardiac transplantation, exercise training increases the capacity for physical work.

Improvements in Lung Function, Exercise, and Quality of Life in Hypercapnic COPD Patients after Lung Volume Reduction Surgery—O'Brien GM, Furukawa S, Kuzma AM, Cordova F, Criner GJ. Chest 1999;115(1):75.

STUDY OBJECTIVE: To determine the impact of preoperative resting hypercapnia on patient outcome after bilateral lung volume reduction surgery (LVRS). METHODS: We prospectively examined morbidity, mortality, quality of life (QOL), and physiologic outcome, including spirometry, gas exchange, and exercise performance in 15 patients with severe em-

physema and a resting P_{aCO_2} of > 45 mm Hg (group 1), and compared the results with those from 31 patients with a P_{aCO_2} of < 45 mm Hg (group 2). RESULTS: All preoperative physiologic and QOL indices were more impaired in the hypercapnic patients than in the eucapnic patients. The hypercapnic patients exhibited a lower preoperative FEV_1, a lower diffusing capacity of the lung for carbon monoxide, a lower ratio of P_{aO_2} to the fraction of inspired oxygen, a lower 6-min walk distance, and higher oxygen requirements. However, after surgery both groups exhibited improvements in FVC (group 1, $p < 0.01$; group 2, $p < 0.001$), FEV_1 (group 1, $p=0.04$; group 2, $p < 0.001$), total lung capacity (TLC; group 1, $p=0.02$; group 2, $p < 0.001$), residual volume (RV: group 1, $p=0.002$; group 2, $p < 0.001$), RV/TLC ratio (group 1, $p=0.03$; group 2, $p < 0.001$), P_{aCO_2} (group 1, $p=0.002$; group 2, $p=0.02$), 6-min walk distance (group 1, $p=0.005$; group 2, $p < 0.001$), oxygen consumption at peak exercise (group 1, $p=0.02$; group 2, $p=0.02$), total exercise time (group 1, $p=0.02$; group 2, $p=0.02$), and the perceived overall QOL scores (group 1, $p=0.001$; group 2, $p < 0.001$). However, because the magnitude of improvement was similar in both groups, and the hypercapnic group was more impaired, the spirometry, lung volumes, and 6-min walk distance remained significantly lower post-LVRS in the hypercapnic patients. There was no difference in mortality between the groups ($p=0.9$). CONCLUSIONS: Patients with moderate to severe resting hypercapnia exhibit significant improvements in spirometry, gas exchange, perceived QOL, and exercise performance after bilateral LVRS. The maximal achievable improvements in postoperative lung function are related to preoperative level of function; however, the magnitude of improvement can be expected to be similar to patients with lower resting P_{aCO_2} levels. Patients should not be excluded from LVRS based solely on the presence of resting hypercapnia. The long-term benefit of LVRS in hypercapnic patient remains to be determined.

Lack of Correlation of Symptoms with Specialist-Assessed Long-Term Asthma Severity—Osborne ML, Vollmer WM, Pedula KL, Wilkins J, Buist AS, O'Hollaren M. Chest 1999; 115(1):85.

STUDY OBJECTIVES: To validate three indicators of asthma severity as defined in the National Asthma Education Program (NAEP) guidelines (ie, frequency of symptoms, degree of airflow obstruction, and frequency of use of oral glucocorticoids), alone and in combination, against severity as assessed by pulmonary specialists provided with 24-month medical chart data. DESIGN: Cross-sectional comparison of questionnaire and clinical-based markers of asthma severity with physician-assessed severity based on chart review. The pulmonologists did not have access to the results of the baseline evaluations when making their severity assessments. SETTING AND PARTICIPANTS: Study participants were 193 asthmatic members (age range, 6 to 55 years) of a large health maintenance organization who underwent a baseline evaluation as part of a separate longitudinal study. This evaluation consisted of spirometry, skin prick testing, and a survey that included questions on symptoms and medication use. The participants in the ancillary study were selected, based on their baseline evaluation, to reflect a broad range of asthma severity. RESULTS: Based on the chart review, 86 of the study subjects (45%) had mild disease, 90 (45%) had moderate disease, and 17 (9%) had severe disease. This physician-assessed severity correlated highly ($p \leq 0.013$) with NAEP-based indices of severity based on oral glucocorticoid use (never, infrequently for attacks, frequently for attacks, and daily use) and on spirometry ($FEV_1 > 80\%$ predicted, 60 to 80% predicted, and <60% predicted). It did not, however, correlate with current asthma symptoms (≤ once/week, 2 to 6 times/week, daily) ($p=0.87$). A composite severity score based on spirometry and the glucocorticoid use data still provided an overall agreement of 63%, with a weighted kappa of 0.40. CONCLUSIONS: While current symptoms are the most important concern of patients with asthma, they reflect the current level of asthma control more than underlying disease severity. Investigators must therefore use caution when comparing groups of patients for whom severity categorization is based largely on symptomatology. This observation, that symptoms alone do not reflect disease severity, becomes even more important as health-care delivery moves closer to protocols/practice guidelines and "best treatment" programs that rely heavily on symptoms to guide subsequent treatment decisions.

Quality of Life in Patients with Obstructive Sleep Apnea: Effect of Nasal Continuous Positive Airway Pressure: A Prospective Study—D'Ambrosio C, Bowman T, Mohsenin V. Chest 1999;115(1):123.

BACKGROUND: Obstructive sleep apnea (OSA) is a common condition and is associated with excessive daytime sleepiness and neuropsychological dysfunction. There is limited evidence on the effect of OSA on the quality of life and its response to nasal continuous positive airway pressure (nCPAP) treatment. STUDY OBJECTIVE: To determine the effect of nCPAP on the quality of life in patients with OSA. DESIGN: Prospective determination of nCPAP effect in a case-series analysis. PATIENTS: We studied 29 patients (23 were male and 6 were female) with a mean (±SE) age of 4.4±2.3 years, a body mass index 36.3±2.0 kg/height m^2, and a diagnosis of OSA with respiratory disturbance index (RDI; apnea/hypopnea) of 77±9 events/h. MEASUREMENTS AND RESULTS: The quality of life was assessed by administering a Medical Outcomes Study Short Form-36 questionnaire before and after 8 weeks of nCPAP therapy in polysomnographically documented OSA. All dimensions of the quality of life were significantly impaired when compared with an age- and gender-matched population, expressed as a percentage of normative data: physical functioning, 75%; vitality, 41%; role functioning (physical, 54%; emotional, 61%; social, 66%); general health, 88%; and mental health, 76%. nCPAP therapy significantly improved the sleep-disordered breathing and sleep fragmentation. The nCPAP level for the group was 9.4±0.7 cm H_2O. Eight weeks of nCPAP therapy improved vitality (75%), social functioning (90%), and mental health (96%). The magnitude of improvement was related to the degree of quality of life impairment prior to treatment, rather than to the severity of disease as measured by the RDI and arousal indices. CONCLUSIONS: All aspects of the quality of life, from physical and emotional health to social functioning, are markedly impaired by OSA. nCPAP therapy improved those aspects related to vitality, social functioning, and mental health.

Noninvasive Positive Pressure Ventilation: Successful Outcome in Patients with Acute Lung Injury/ARDS—Rocker GM, Mackenzie MG, Williams B, Logan PM. Chest 1999;115(1): 173.

BACKGROUND: There is increasing support for the use of noninvasive positive pressure ventilation (NPPV) in the treatment of patients with acute respiratory failure. Highest success rates are recorded in patients with exacerbation of COPD, particularly in patients presenting primarily with hypercarbic respiratory failure. Success has been more limited in patients with acute hypoxemic respiratory failure, and there are few reports of NPPV in patients with acute lung injury (ALI) or ARDS. OBJECTIVES: We report the outcome of 12 episodes of ALI/ARDS in 10 patients treated with NPPV. DESIGN: Experiential cohort study. SETTING: Tertiary referral center and university hospital ICU. INTERVENTION: Provision of NPPV in patients with ALI/ARDS. RESULTS: Group median (range) APACHE (acute physiology and chronic health evaluation) II score was 16 (11 to 29). Success rate (avoidance of intubation and no further assisted ventilation for 72 h) was achieved on six of nine occasions (66%) when NPPV was used as the initial mode of assisted ventilation. It failed after three episodes of planned (1) or self (2) extubation. Duration of successful NPPV was 64.5 h (23.5 to 80.5 h) with ICU discharge in the next 24 to 48 h for three of six patients. Unsuccessful episodes lasted 7.3 h (0.1 to 116 h) with need for conventional ventilation for an additional 5 days

(2.7 to 14 days). Survival (ICU and hospital) for the 10 patients was 70%. CONCLUSIONS: In a group of hemodynamically stable patients with severe ALI, NPPV had a high success rate. NPPV should be considered as a treatment option for patients in stable condition in the early phase of ALI/ARDS.

The Effect of Heliox on Nebulizer Function Using a Beta-Agonist Bronchodilator—Hess DR, Acosta FL, Ritz RH, Kacmarek RM, Camargo CA Jr. Chest 1999;115(1):184.

OBJECTIVE: To evaluate nebulizer performance when heliox was used to power the nebulizer. METHODS: Conventional and continuous nebulizer designs were evaluated. The conventional nebulizer was used with 5 mg albuterol and flows of 8 L/min air, 8 L/min heliox, and 11 L/min heliox; it was also used with 10 mg albuterol and a heliox flow of 8 L/min. The continuous nebulizer was set to deliver 10 mg of albuterol over 40 min at flows of 2 L/min air, 2 L/min heliox, and 3 L/min heliox; it was also used with 20 mg albuterol and a heliox flow of 2 L/min. A cotton plug at the nebulizer mouthpiece was used to trap aerosol during simulated spontaneous breathing. The amount of albuterol deposited on the cotton plug was determined spectrophotometrically. Particle size was determined using an 11-stage cascade impactor. RESULTS: For both nebulizer designs, particle size and inhaled mass of albuterol decreased significantly ($p < 0.001$) when the nebulizer was powered with heliox rather than air. When powered with heliox, the reduction in inhaled mass of albuterol was less for the conventional nebulizer (16%) than the continuous nebulizer (67%). The nebulization time, however, was more than twofold greater with heliox ($p < 0.001$). Increasing the flow of heliox increased the particle size ($p < 0.05$), inhaled mass of albuterol ($p < 0.05$), and inhaled mass of particles 1 to 5 microm ($p < 0.05$) to levels similar to powering the nebulizer with air at the lower flow. Increasing the albuterol concentration in the nebulizer and using the lower heliox flow increased the inhaled mass of albuterol ($p < 0.05$) while maintaining the smaller particle size produced with that flow. CONCLUSIONS: The use of heliox to power a nebulizer affects both the inhaled mass of medication and the size of the aerosol particles. The flow to power the nebulizer should be increased when heliox is used.

Reduced Quality of Life In Survivors of Acute Respiratory Distress Syndrome Compared with Critically Ill Control Patients—Davidson TA, Caldwell ES, Curtis JR, Hudson LD, Steinberg KP. JAMA 1999;281(4):354.

CONTEXT: Health-related quality of life (HRQL) is reduced in patients who survive acute respiratory distress (ARDS), but whether this decline in HRQL is caused by ARDS or other aspects of the patient's illness or injury is unknown. OBJECTIVE: To determine if there are differences in the HRQL of ARDS survivors and comparably ill or injured controls without ARDS. DESIGN: Prospective, matched, parallel cohort study. SETTING: A 411-bed municipal medical and regional level I trauma center. PATIENTS: Seventy-three pairs of ARDS survivors and severity-matched controls with the clinical risk factors for ARDS of sepsis and trauma admitted between January 1, 1994, and July 30, 1996. MAIN OUTCOME MEASURES: The HRQL of ARDS survivors and controls, assessed by generic and pulmonary disease-specific HRQL instruments (Medical Outcomes Study 36-Item Short Form Health Survey, Standard Form [SF-36] and St George's Respiratory Questionnaire [SGRQ], respectively). RESULTS: Clinically meaningful and statistically significant reductions in HRQL scores of ARDS survivors ($n = 73$) were seen in 7 of 8 SF-36 domains and 3 of 3 SGRQ domains compared with matched controls ($P < 0.001$ for all reductions). The largest decrements in the HRQL were seen in physical function and pulmonary symptoms and limitations. Analysis of trauma-matched pairs ($n = 46$) revealed significant reductions in 7 of 8 SF-36 domains ($P \leq 0.02$) and 3 of 3 SGRQ domains ($P \leq 0.003$). Analysis of sepsis-matched pairs ($n = 27$) revealed significant reductions in 6 of 8 SF-36 domains ($P \leq 0.05$) and 3 of 3 SGRQ domains ($P \leq 0.002$). CONCLUSIONS: Survivors of ARDS have a clinically significant reduction in HRQL that appears to be caused exclusively by ARDS and its sequelae. Reductions were primarily noted in physical functioning and pulmonary disease-specific domains.

Acquired Neuromuscular Disorders in Critically Ill Patients: A Systematic Review. Groupe de Reflexion et d'Etude sur les Neuromyopathies En Reanimation—De Jonghe B, Cook D, Sharshar T, Lefaucheur JP, Carlet J, Outin H. Intensive Care Med 1998;24(12):1242.

OBJECTIVE: To summarize the prospective clinical studies of neuromuscular abnormalities in intensive care unit (ICU) patients. STUDY IDENTIFICATION AND SELECTION: Studies were identified through MEDLINE, EMBASE, references in primary and review articles, personal files, and contact with authors. Through duplicate independent review, we selected prospective cohort studies evaluating ICU-acquired neuromuscular disorders. DATA ABSTRACTION: In duplicate, independently, we abstracted key data regarding design features, the population, clinical and laboratory diagnostic tests, and clinical outcomes. RESULTS: We identified eight studies that enrolled 242 patients. Inception cohorts varied; some were mechanically ventilated patients for ≥ 5 days, others were based on a diagnosis of sepsis, organ failure, or severe asthma while others were selected on the basis of exposure to muscle relaxants, or because of participation in muscle biochemistry studies. Weakness was systematically assessed in two of the eight studies, concerning patients with severe asthma, with a reported frequency of 36 and 70%, respectively. Electrophysiologic and histologic abnormalities consisted of both peripheral nerve and muscle involvement and were frequently reported, even in non-selected ICU patients. In a population of patients mechanically ventilated for more than 5 days, electrophysiologic abnormalities were reported in 76% of cases. Two studies showed a clinically important increase (5 and 9 days, respectively) in duration of mechanical ventilation and a mortality twice as high in patients with critical illness neuromuscular abnormalities, compared to those without. CONCLUSIONS: Prospective studies of ICU-acquired neuromuscular abnormalities include a small number of patients with various electrophysiologic findings but insufficiently reported clinical correlations. Evaluation of risk factors for these disorders and studies examining their contribution to weaning difficulties and long-term disability are needed.

Quality of Survival after Cardiopulmonary Resuscitation—de Vos R, de Haes HC, Koster RW, de Haan RJ. Arch Intern Med 1999;159(3):249.

BACKGROUND: Outcome of cardiopulmonary resuscitation (CPR) can be poor, in terms of life expectancy and quality of life. OBJECTIVES: To determine the impact of patient characteristics before, during, and after CPR on these outcomes, and to compare results of the quality-of-life assessment with published studies. METHODS: In a cohort study, we assessed by formal instruments the quality of life, cognitive functioning, depression, and level of dependence of survivors after inhospital CPR. Follow-up was at least 3 months after discharge from the hospital (tertiary care center). RESULTS: Of 827 resuscitated patients, 12% ($n = 101$) survived to follow-up. Of the survivors, 89% participated in the study. Most survivors were independent in daily life (75%), 17% were cognitively impaired, and 16% had depressive symptoms. Multivariate regression analysis showed that quality of life and cognitive function were determined by 2 factors known before CPR-the reason for admission and age. Factors during and after resuscitation, such as prolonged cardiac arrest and coma, did not significantly determine the quality of life or cognitive functioning of survivors. The quality of life of our CPR survivors was worse compared with a reference group of elderly individuals, but better than that of a reference group of patients with stroke. The quality of life did not importantly differ between the compared studies of CPR survivors. CONCLUSIONS: Car-

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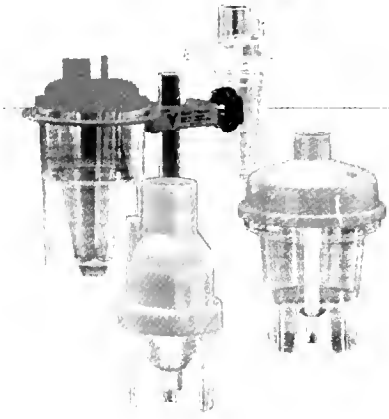

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diopulmonary resuscitation is frequently unsuccessful, but if survival is achieved, a relatively good quality of life can be expected. Quality of life after CPR is mostly determined by factors known before CPR. These findings may be helpful in informing patients about the outcomes of CPR.

Contaminated Aerosol Recovery from Pulmonary Function Testing Equipment—Hiebert T, Miles J, Okeson GC. Am J Respir Crit Care Med 1999;159(2):610.

Clinically, the spread of infectious agents between subjects undergoing spirometry is quite uncommon. There is almost no documentation in the medical literature on this subject. We studied the retrieval of nonpathogenic Escherichia coli after aerosolizing organisms into standard pulmonary function tubing of a type that is frequently used by volume-sensing spirometers. The arrival of the aerosol at the distal end of the tubing was documented by culture. After delays of 0, 1, 5, and 10 min, respectively, air was forcibly withdrawn from the proximal end of the tubing through a special petri plate assembly. The plates were cultured and the colonies were counted. Immediately after insufflation of organisms, air withdrawn from the proximal tubing had counts similar to the air sampled at the distal end. After a 1-min delay, the proximal samples contained only rare organisms. No organisms were recovered from proximal air samples after a delay of 5 or 10 min after insufflation of organisms. The absence of detectable aerosolized E. coli after delays of 5 and 10 min after insufflation of organisms into spirometry tubing supports the hypothesis that a significant transfer of aerosolized organisms does not occur during routine pulmonary function testing as long as an interval of 5 min or more is allowed between tests.

Effect of Spontaneous Breathing Trial Duration on Outcome of Attempts to Discontinue Mechanical Ventilation—Esteban A, Alia I, Tobin MJ, Gil A, Gordo F, Vallverdu I, et al. Am J Respir Crit Care Med 1999;159(2):512.

The duration of spontaneous breathing trials before extubation has been set at 2 h in research studies, but the optimal duration is not known. We conducted a prospective, multicenter study involving 526 ventilator-supported patients considered ready for weaning, to compare clinical outcomes for trials of spontaneous breathing with target durations of 30 and 120 min. Of the 270 and 256 patients in the 30- and 120-min trial groups, respectively, 237 (87.8%) and 216 (84.8%), respectively, completed the trial without distress and were extubated ($p = 0.32$); 32 (13.5%) and 29 (13.4%), respectively, of these patients required reintubation within 48 h. The percentage of patients who remained extubated for 48 h after a spontaneous breathing trial did not differ in the 30- and 120-min trial groups (75.9% versus 73.0%, respectively, $p = 0.43$). The 30- and 120-min trial groups had similar within-unit mortality rates (13 and 9%, respectively) and in-hospital mortality rates (19 and 18%, respectively). Reintubation was required in 61 (13.5%) patients, and these patients had a higher mortality (20 of 61, 32.8%) than did patients who tolerated extubation (18 of 392, 4.6%) ($p < 0.001$). Neither measurements of respiratory frequency, heart rate, systolic blood pressure, and oxygen saturation during the trial, nor other functional measurements before the trial discriminated between patients who required reintubation from those who tolerated extubation. In conclusion, after a first trial of spontaneous breathing, successful extubation was achieved equally effectively with trials targeted to last 30 and 120 min.

Changes in the Work of Breathing Induced by Tracheotomy in Ventilator-Dependent Patients—Diehl JL, Atrous SE, Touchard D, Lemaire F, Brochard L. Am J Respir Crit Care Med 1999;159(2):383.

Tracheotomy is widely performed on ventilator-dependent patients, but its effects on respiratory mechanics have not been studied. We measured the work of breathing (WOB) in eight patients before and after tracheotomy during breathing at three identical levels of pressure support (PS): baseline level (PS-B), PS + 5 cm H_2O (PS+5), and PS - 5 cm H_2O (PS-5). After the procedure, we also compared the resistive work induced by the patients' endotracheal tubes (ETTs) and by a new tracheotomy cannula in an in vitro bench study. A significant reduction in the WOB was observed after tracheotomy for PS-B (from 0.9 ± 0.4 to 0.4 ± 0.2 J/L, $p < 0.05$), and for PS-5 (1.4 ± 0.6 to 0.6 ± 0.3 J/L, $p < 0.05$), with a near-significant reduction for PS+5 (0.5 ± 0.5 to 0.2 ± 0.1 J/L, $p = 0.05$). A significant reduction was also observed in the pressure-time index of the respiratory muscles (181 ± 92 to 80 ± 56 cm $H_2O \cdot$ s/min for PS-B, $p < 0.05$). Resistive and elastic work computed from transpulmonary pressure measurements decreased significantly at PS-B and PS-5. A significant reduction in occlusion pressure and intrinsic positive end-expiratory pressure (PEEP) was also observed for all conditions, with no significant change in breathing pattern. Three patients had ineffective breathing efforts before tracheotomy, and all had improved synchrony with the ventilator after the procedure. In vitro measurements made with ETTs removed from the patients, with new ETTs, and with the tracheotomy cannula showed that the cannula reduced the resistive work induced by the artificial airway. Part of these results was explained by a slight, subtle reduction of the inner diameter of used ETTs. We conclude that tracheotomy can substantially reduce the mechanical workload of ventilator-dependent patients.

Accuracy of Pulse Oximetry in Sickle Cell Disease—Ortiz FO, Aldrich TK, Nagel RL, Benjamin LJ. Am J Respir Crit Care Med 1999; 159(2):447.

Pulmonary complications and hypoxemia are common in sickle cell disease (SCD) and may exacerbate microvascular occlusive phenomena. Thus, detecting hypoxemia is of particular importance in SCD. To assess the accuracy of pulse oximetry in the diagnosis of hypoxemia in SCD, we compared 22 pulse oximetric measurements of arterial oxygen saturation (S_{pO_2}) in adult patients with SCD and acute vasoocclusive crisis with simultaneously drawn arterial saturation (S_{aO_2}2 = oxyhemoglobin divided by oxyhemoglobin plus reduced hemoglobin) measured by co-oximetry. We accepted S_{pO_2} readings only if they were stable and characterized by strong and regular photoplethysmographic waves on the oximeter screen. To assess the position of these patients' oxyhemoglobin dissociation curves, we plotted arterial and venous oxygen saturation (S_{aO_2} and S_{vO_2}) against oxygen tension. We found right-shifted oxyhemoglobin dissociation curves, with pH-corrected p50s ranging from 28 to 38 mm Hg. Pulse oximetry slightly overestimated oxyhemoglobin percentage (by an average of 3.4 percentage points), but it almost always accurately estimated S_{aO_2} (underestimating on average by 1.1 percentage points). The error in S_{pO_2} was never enough to classify a hypoxemic patient erroneously as normoxemic or a normoxemic patient as hypoxemic. We conclude that, as long as strong and regular photoplethysmographic waves are present, pulse oximeters can be relied upon not to misdiagnose either hypoxemia or normoxemia in SCD.

As-Required Versus Regular Nebulized Salbutamol for the Treatment of Acute Severe Asthma—Bradding P, Rushby I, Scullion J, Morgan MD. Eur Respir J 1999;13(2):290.

Current British guidelines for the administration of β2-agonists in acute severe asthma recommend regular nebulized therapy in hospitalized patients, followed by as-required (p.r.n.) use via hand-held devices after discharge. Since β2-agonists do not possess anti-inflammatory activity in vivo, and are thus unlikely to influence the rate of recovery from an asthma exacerbation, it was hypothesized that patients given the short-acting β2-agonist salbutamol on an as-required basis after admission to hospital would recover as quickly as those on regular treatment, but with potential reductions in the total dose delivered. Forty-six patients with acute severe asthma were randomly assigned to either regular prescriptions of nebulized salbutamol or to usage on a p.r.n. basis, from 24 h

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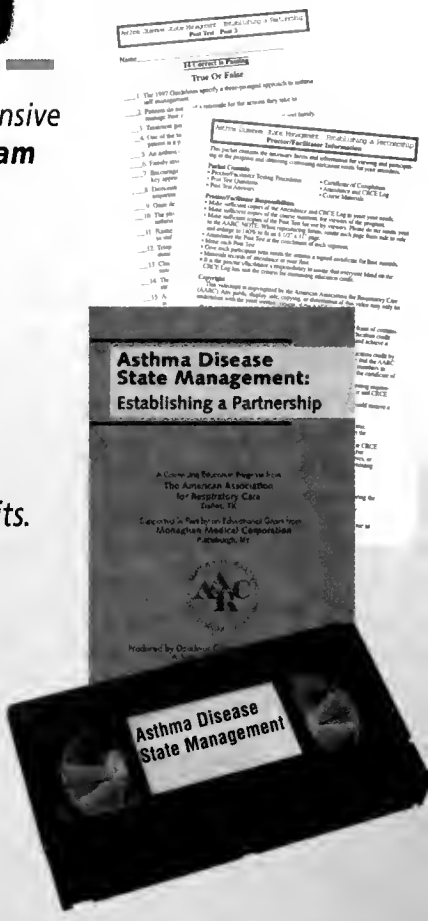

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after hospital admission. The primary outcome measures were length of hospital stay, time to recovery, and frequency of salbutamol nebulization from 24 h after admission to discharge. Secondary outcome measures were treatment side-effects (tremor, palpitations), and patient satisfaction. Length of hospital stay was reduced in those patients allocated to p.r.n. salbutamol (geometric mean (GM) 3.7 days) versus regular salbutamol (GM 4.7 days). Time taken for peak expiratory flow to reach 75% of recent best was the same in both groups. There was a highly significant reduction in the number of times nebulized therapy was delivered to the p.r.n. group (GM 7.0, range 1–30) compared with the regular treatment group (GM 14.0, range 4–57; p=0.003; 95% confidence interval for ratio of GMs 1.29–3.09). In addition, patients reported less tremor (p=0.062) and fewer palpitations (p=0.049) in the p.r.n. group. Of the patients in the p.r.n. group who had received regular nebulized therapy on previous admissions (n=12), all preferred the p.r.n. regimen. Prescribing β2-agonists on a p.r.n. basis from 24 h after hospital admission is associated with reduced amount of drug delivered, incidence of side-effects, and possibly length of hospital stay. This has implications for the efficient use of healthcare resources.

Human Lung Volumes and the Mechanisms that Set Them—Leith DE, Brown R. Eur Respir J 1999;13(2):468.

Definitions of human lung volumes and the mechanisms that set them are reviewed in the context of pulmonary function testing, with attention to the distinction between functional residual capacity (FRC) and the static relaxation volume of the respiratory system, and to the circumstances in which FRC and residual volume are set by dynamic rather than by static mechanisms. Related terms, conventions, and issues are addressed, including some common semantic and conceptual difficulties, with attention to "gas trapping", "hyperinflation", and "restriction".

Delays in the Suspicion and Treatment of Tuberculosis Among Hospitalized Patients—Rao VK, Iademarco EP, Fraser VJ, Kollef MH. Ann Intern Med 1999;130(5):404.

BACKGROUND: Despite increased awareness of tuberculosis, delays in management are common. OBJECTIVE: To investigate management delays among hospitalized patients with tuberculosis. DESIGN: Retrospective cohort study. SETTING: The Barnes-Jewish-Christian Health System, a network of eight community and tertiary-care facilities serving the St. Louis, Missouri, metropolitan area. PATIENTS: All 203 patients with tuberculosis hospitalized in the Barnes-Jewish-Christian Health System from 1988 to 1996. MEASUREMENTS: Time from admission to first consideration of the diagnosis (suspicion interval), first consideration and treatment initiation (treatment interval), and admission and treatment initiation (overall management interval) were determined. Delays were defined as intervals longer than 24 hours. RESULTS: The overall management interval (median, 6 days [5th and 95th percentiles, 1 and 52 days]) exceeded 24 hours in 152 patients (74.9% [95% CI, 68.9% to 80.9%]). The suspicion interval (median, 1 day [5th and 95th percentiles, 0 and 16 days]) exceeded 24 hours in 54 patients (26.6% [CI, 20.5% to 32.7%]), and the treatment interval (median, 3 days [5th and 95th percentiles, 0 and 51 days]) was prolonged in 130 patients (64.0% [CI, 57.4% to 70.6%]). Overall management delays of more than 10 and 25 days occurred in 33.5% (CI, 27.0% to 40.0%) and 18.7% (CI, 13.3% to 24.1%) of patients, respectively. The 55 patients with smears that were positive for acid-fast bacilli had a median treatment interval of 3 days (5th and 95th percentiles, 0 and 33 days); in 58.2% of patients (CI, 45.2% to 71.2%), this interval exceeded 24 hours. CONCLUSIONS: Delays in initiation of treatment were more common than delays in the initial suspicion of tuberculosis. Both types of delays were common even in patients with disease that was confirmed by a positive smear. These data illustrate a need for improved education of physicians about the benefits of early initiation of therapy for tuberculosis.

A Controlled Trial of Sustained-Release Bupropion, a Nicotine Patch, or Both for Smoking Cessation—Jorenby DE, Leischow SJ, Nides MA, Rennard SI, Johnston JA, Hughes AR, et al. N Engl J Med 1999;340(9):685.

BACKGROUND AND METHODS: Use of nicotine-replacement therapies and the antidepressant bupropion helps people stop smoking. We conducted a double-blind, placebo-controlled comparison of sustained-release bupropion (244 subjects), a nicotine patch (244 subjects), bupropion and a nicotine patch (245 subjects), and placebo (160 subjects) for smoking cessation. Smokers with clinical depression were excluded. Treatment consisted of nine weeks of bupropion (150 mg a day for the first three days, and then 150 mg twice daily) or placebo, as well as eight weeks of nicotine-patch therapy (21 mg per day during weeks 2 through 7, 14 mg per day during week 8, and 7 mg per day during week 9) or placebo. The target day for quitting smoking was usually day 8. RESULTS: The abstinence rates at 12 months were 15.6 percent in the placebo group, as compared with 16.4 percent in the nicotine-patch group, 30.3 percent in the bupropion group (P<0.001), and 35.5 percent in the group given bupropion and the nicotine patch (P<0.001). By week 7, subjects in the placebo group had gained an average of 2.1 kg, as compared with a gain of 1.6 kg in the nicotine-patch group, a gain of 1.7 kg in the bupropion group, and a gain of 1.1 kg in the combined-treatment group (P<0.05). Weight gain at seven weeks was significantly less in the combined-treatment group than in the bupropion group and the placebo group (P<0.05 for both comparisons). A total of 311 subjects (34.8 percent) discontinued one or both medications. Seventy-nine subjects stopped treatment because of adverse events: 6 in the placebo group (3.8 percent), 16 in the nicotine-patch group (6.6 percent), 29 in the bupropion group (11.9 percent), and 28 in the combined-treatment group (11.4 percent). The most common adverse events were insomnia and headache. CONCLUSIONS: Treatment with sustained-release bupropion alone or in combination with a nicotine patch resulted in significantly higher long-term rates of smoking cessation than use of either the nicotine patch alone or placebo. Abstinence rates were higher with combination therapy than with bupropion alone, but the difference was not statistically significant.

Effect of Oxygen Treatment on Heart Rate after Abdominal Surgery—Rosenberg-Adamsen S, Lie C, Bernhard A, Kehlet H, Rosenberg J. Anesthesiology 1999;90(2):380.

BACKGROUND: Cardiac complications are common during the postoperative period and may be associated with hypoxemia and tachycardia. Preliminary studies in high-risk patients after operation have shown a possible beneficial effect of oxygen therapy on arterial oxygen saturation and heart rate. METHODS: The authors studied the effect of oxygen therapy on arterial oxygen saturation and heart rate in 100 consecutive unselected patients randomly and double blindly allocated to receive air or oxygen therapy between the first and fourth day after major abdominal surgery. RESULTS: The median arterial oxygen saturation rate increased significantly from 96% to 99% (P < 0.0001) and the heart rate decreased significantly from 85 beats/min to 81 beats/min (P < 0.0001) during oxygen supplementation compared with air administered by a binasal catheter. The greatest decrease in heart rate occurred in patients with the lowest oxygen saturation or the highest heart rate values before oxygen supplementation. Overall, 73% of this unselected group of patients responded with decreased heart rate during supplemental oxygen therapy. No significant differences in changes in heart rate after oxygen supplementation were found between patients with or without an epidural catheter or between the postoperative day studied. CONCLUSION: Postoperative oxygen therapy increased arterial oxygen saturation and decreased heart rate after uncomplicated abdominal surgery in a consecutive unselected group of patients who received routine postoperative care.

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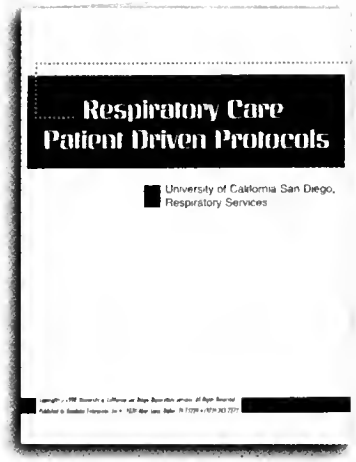

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Endotracheal Intubation, but Not Laryngeal Mask Airway Insertion, Produces Reversible Bronchoconstriction—Kim ES, Bishop MJ. Anesthesiology 1999;90(2):391.

BACKGROUND: Tracheal intubation frequently results in an increase in respiratory system resistance that can be reversed by inhaled bronchodilators. The authors hypothesized that insertion of a laryngeal mask airway would be less likely to result in reversible bronchoconstriction than would insertion of an endotracheal tube. METHODS: Fifty-two (45 men, 7 women) patients were randomized to receive a 7.5-mm (women) or 8-mm (men) endotracheal tube or a No. 4 (women) or No. 5 (men) laryngeal mask airway. Anesthesia was induced with 2 microg/kg fentanyl and 5 mg/kg thiopental, and airway placement was facilitated with 1 mg/kg succinylcholine. When a seal to more than 20 cm water was verified, respiratory system resistance was measured immediately after airway placement. Inhalation anesthesia was begun with isoflurane to achieve an end-tidal concentration of 1% for 10 min. Respiratory system resistance was measured again during identical conditions. RESULTS: Among patients receiving laryngeal mask airways, the initial respiratory system resistance was significantly less than among patients with endotracheal tubes (9.2±3.3 cm water x l^{-1} x s^{-1} [mean ± SD] compared with 13.4±9.6 cm water x l^{-1} x s^{-1}; $P < 0.05$). After 10 min of isoflurane, the resistance decreased to 8.6±3.6 cm water x l^{-1} x s^{-1} in the endotracheal tube group but remained unchanged at 9.1±3.3 cm water x l^{-1} x s^{-1} in the laryngeal mask airway group. The decrease in respiratory system resistance in the endotracheal tube group of 4.7±7 cm water x l^{-1} x s^{-1} was highly significant compared with the lack of change in the laryngeal mask airway group ($P < 0.01$). CONCLUSIONS: Resistance decreased rapidly only in patients with endotracheal tubes after they received isoflurane, a potent bronchodilator, suggesting that reversible bronchoconstriction was present in patients with endotracheal tubes but not in those with laryngeal mask airways. A laryngeal mask airway is a better choice of airway to minimize airway reaction.

Influence of Pulse Oximetry and Capnography on Time to Diagnosis of Critical Incidents in Anesthesia: A Pilot Study Using a Full-Scale Patient Simulator—Lampotang S, Gravenstein JS, Euliano TY, van Meurs WL, Good ML, Kubilis P, Westhorpe R. J Clin Monit Comput 1998;14(5):313.

OBJECTIVE: Many studies (outcome, epidemiological) have tested the hypothesis that pulse oximetry and capnography affect the outcome of anesthetic care. Uncontrollable variables in clinical studies make it difficult to generate statistically conclusive data. In the present study, we eliminated the variability among patients and operative procedures by using a full-scale patient simulator. We tested the hypothesis that pulse oximetry and capnography shorten the time to diagnosis of critical incidents. METHODS: A simulator was programmed to represent a patient undergoing medullary nailing of a fractured femur under general anesthesia and suffering either malignant hyperthermia, a pneumothorax, a pulmonary embolism or an anoxic oxygen supply. One hundred thirteen anesthesiologists were randomly assigned to one of two groups of equal size, one with access to pulse oximetry and capnography data and the other without. Each anesthesiologist was further randomized to one of the four critical incidents. Each anesthetic procedure was videotaped. The time to correct diagnosis was measured and analyzed. RESULTS: Based on analysis of 91 of the subjects, time to diagnosis was significantly shorter (median of 432 s vs. >480 s) for the anoxic oxygen supply scenario ($p = 0.019$) with pulse oximetry and capnography than without. No statistical difference in time to diagnosis was obtained between groups for the other three critical incidents. CONCLUSIONS: Simulation may offer new approaches to the study of monitoring technology. However, the limitations of current simulators and the resources required to perform simulator-based research are impediments to wide-spread use of this tool.

Evaluation of Volunteers of the VIA V-ABG Automated Bedside Blood Gas, Chemistry, and Hematocrit Monitor—Bailey PL, McJames SW, Cluff ML, Wells DT, Orr JA, Westenskow DR, Kern SE. J Clin Monit Comput 1998;14(5):339.

OBJECTIVE: To evaluate the VIA V-ABG (VIA Medical Corp.) point-of-care bloodgas and chemistry monitor in healthy human volunteers, with particular emphasis on the measurement of blood gases. METHODS: Experimental conditions were varied by intermittently subjecting volunteers to either isocapnic hypercapnia (end-tidal (ET), $PETCO_2$ = 50±2 mmHg, $ETPO_2$ = 130±5 mmHg) or isocapnic hypoxia ($PETCO_2$ = 42±2, $PETO_2$ + 45±2 mmHg) in addition to room air breathing. Measurements by the VIA V-ABG device were compared with paired samples and measurements performed by two ABL Radiometers (505 and 500). Analysis of results includes bias and precision plots and comparison of results with minimal performance criteria as established by CLIA. RESULTS: Nineteen volunteers yielded 222 matched samples. The range of values were 7.32–7.61 for pH, 20.9–51.6 mmHg for PCO_2, 27.9–184.5 mmHg for PO_2, 134–141 mmol/l for Na, 3.1–4.1 mmol/l for K, and 30.0–50.4% for hematocrit. Bias and precision (±2 sd) for pH was 0.01 and 0.04, for PCO_2 was 0.4 and 4.8, for PO_2 was 1.0 and 17.0, for Na was -0.3 and 5.2, for K was 0.1 and 0.2, and for Hct was 2.0 and 5.4. CONCLUSIONS: Over the range of blood gas values assessed, blood gas measurements by the VIA V-ABG device were clinically acceptable and met minimal performance criteria utilizing current Medicare CLIA proficiency standards. Performance criteria were also met by the VIA V-ABG device for Na, K, and Hct measurements but the range of values was too narrow to allow characterization of clinical acceptability. The VIA V-ABG device appears to perform well compared with the results which have been published for other point-of-care devices. Comparison between different studies investigating point-of-care devices is difficult due to several factors (range of values measured, comparison device, population studied, etc.). Some of these instruments, including the VIA V-ABG device, may serve quite well as point-of-care devices to perform certain tests at the bedside. Whether or not any of these devices can substitute for traditional laboratory blood gas and chemistry measurements remains an issue that is not adequately studied.

Hypokalemia and Salbutamol Therapy in Asthma—Hung CH, Chu DM, Wang CL, Yang KD. Pediatr Pulmonol 1999;27(1):27.

Hypokalemia is a common side effect in adult asthmatic patients on β 2 adrenergic therapy. There is limited information in regard to hypokalemia and its relation to the clinical responses following administration of β 2 agonist therapy in children with asthma. We observed that salbutamol inhalation significantly improved asthmatic symptoms as demonstrated by increases in peak expiratory flow (PEF: 122.37±75.38 vs. 152.59±80.29; $P < 0.001$) and venous oxygen tension (Pv,O_2: 33.24±4.95 vs. 58.16±2.31; $P < 0.001$), and decreases in respiratory rate (RR: 36.39±3.78 vs. 28.62±3.12; $P < 0.01$), clinical scores (CS: 3.59±1.28 vs. 1.59±0.71), and venous PCO_2 tensions ($PvCO_2$: 40.84±2.67 vs. 34.75±2.31; $P < 0.001$). Salbutamol-induced hypokalemia was correlated with a decrease in RR, and an increase of PvO_2 and PEF. These findings suggest that the same mechanism is involved in eliciting hypokalemia and bronchodilatation.

Can We Rehabilitate the Chest Wall?

Thomas A Dillard MD

Stedman's Medical Dictionary defines "rehabilitation" as restoration of the ability to function in a normal or near normal manner following disease, illness, or injury.[1] As noted in a recent review, the primary goals of pulmonary rehabilitation programs are to relieve symptoms, particularly dyspnea, improve functional ability, and enhance health-related quality of life.[2] New modalities of therapy that may benefit patients in pulmonary rehabilitation programs are emerging, and this trend reflects increasing scientific knowledge and technology as well as the gradual emergence and acceptance of this application of rehabilitation.

In this issue of RESPIRATORY CARE, Kakizaki et al's report on a potentially useful new pulmonary rehabilitation modality, "respiratory stretch gymnastics."[3] This article has merit for several reasons, including input from the perspective of physical therapists and physiologists as well as rehabilitation and pulmonary clinicians, input from an international contributor, and, perhaps most importantly, suggestion of a novel topic for further study and possibly a clinical breakthrough for pulmonary rehabilitation patients.

SEE THE ORIGINAL STUDY ON PAGE 409

Kakizaki et al prospectively evaluated the chest circumference, Fletcher's dyspnea classification, and vital capacity of 22 patients with chronic obstructive pulmonary disease before and after instruction in 5 stretching techniques intended to stretch the rib cage, shoulders, thoracic spine, and cervical spine. Patients were instructed to perform stretching 3 times a day for 4 weeks. The main results include: an increase in upper chest wall expansion and reduction, and smaller changes in the lower chest wall; improved dyspnea rating in 12 of 22 patients; and increased vital capacity. The patients with the greatest dyspnea ratings appeared to be the most likely to respond. These findings, and the proposed mechanism of improvement, pose a serious and interesting question for further study, and the results might help determine the appropriate sample size for future studies. If properly studied and validated in the future, implementation of these stretching techniques may benefit patients in pulmonary rehabilitation programs.

The novelty of the report is in viewing the chest wall as a target of opportunity for pulmonary rehabilitation. The chest wall may well be the source of morbidity for patients with congenital or other deformities such as kyphoscoliosis. Begin et al previously reported reduced compliance of the rib cage in patients with rheumatoid arthritis (compared to normal controls), which may contribute to reduced lung volume.[4] Can the aging chest wall contribute to morbidity in chronic obstructive pulmonary disease? The muscles of the chest wall and diaphragm, and the accessory respiratory muscles have for years been an important topic of pulmonary rehabilitation research, so it is an intriguing hypothesis that stretching the elastic elements of the chest wall can improve function and quality of life.

The report by Kakizaki et al does have serious limitations, however, and, as the study's title suggests, should be considered a preliminary report. I would classify the report as hypothesis-generating rather than hypothesis-confirming, by which I mean the study does not definitively confirm the hypothesis that the stretching changes the elastic properties of the chest wall. Moreover, the considerable limitations of the methods of study suggest caution in interpreting the results, regardless of the mechanism. At present, I strongly favor further study instead of immediate implementation.

The design of the study could be greatly strengthened by randomization to a treated or control population, and by blinding the study to both the patients and therapists. Such a design would minimize the bias of ascertainment, the natural tendency to affirm the question under investigation. Another suggestion would be to establish variability in patient measurements and to document stability over several weeks before beginning interventions. This would remove the possibility of recruiting patients who are in the improving phase after an exacerbation. Measurements should certainly be obtained close to the intervention in this population, noted for frequent exacerbations.

Kakizaki et al objectively assessed patient adherence to the training regimen one time only, by observing how well the patients could perform the stretching techniques after the intended 4-week training period. This approach ap-

pears sound, but I would favor earlier or serial objective monitoring, review of a patient diary or other log, by having the patients come to the clinic for some of the sets of stretching. It is a bit disappointing that 12 patients were rated as having poor performance of the stretch techniques after 4 weeks.

The measured parameters could also be improved. The Fletcher dyspnea scale measures dyspnea status with 5 possible discrete choices. Perhaps a more continuous and less discrete scale, such as a visual analog scale or other scale with more choices, would allow detection of subtler changes. Evaluating change in dyspnea or dyspnea during a fixed challenge, in addition to current dyspnea status, might be more convincing. A further consideration concerning dyspnea is the possible effect of the stretching techniques to increase upper extremity strength or fitness. Patients with severe chronic obstructive pulmonary disease report a marked increase in the sensation of dyspnea with unsupported arm elevation.[5] Because the stretching regimen involves upper extremity maneuvers, dyspnea may have improved due to training of the upper extremities. A quantitative measure of upper extremity strength or fitness might be necessary to control for this possibility.

A final consideration on the topic of dyspnea involves the role of chest wall afferent nerve firing. A previous study using in-phase chest wall vibration suggested that effects on respiratory sensation are mediated by afferent information from chest wall respiratory muscles.[6] It would be very interesting to find out whether stretching the chest wall could change afferent information, and how long this might last; however, such an experiment might require animal preparations to be conclusive. If the afferent mechanoreceptor mechanism is at work, then this mechanism may not require a change in chest wall mechanics to benefit the patient.

Kakizaki et al speculate that the stretching techniques may decrease elastance (increase compliance) of the chest wall. Confirming this hypothesis would be worthwhile, but difficult. Elastance, defined as pressure change over volume change ($E = \Delta P/\Delta V$) is the reciprocal of compliance. That is, elastance represents "stiffness." Compliance of the total respiratory system, lung, and chest wall is familiar to all of us from monitoring ventilated patients.

To obtain a measurement of chest wall elastance requires partitioning of the total respiratory system elastance into its 2 components: lung and chest wall. To do this in mechanically ventilated or spontaneously breathing patients requires obtaining an estimate of pleural pressure, such as esophageal pressure. Fortunately, the techniques for measuring lung mechanics, including static and dynamic chest wall elastance, have recently been reinvigorated by studies involving lung volume reduction surgery.[7] It will be interesting to learn if stretch techniques produce an improvement in chest wall elastance.

It may be that the stretch techniques increase the range of motion, or maximal extent of displacement, without an associated change in $\Delta P/\Delta V$. This would manifest as an increase in the maximal pressure at total lung capacity along with an increase in vital capacity. Looking for this effect would also require an estimate of esophageal pressure. To fully evaluate the physiologic effects of repeated stretching, it also seems appropriate to measure respiratory muscle strength by maximal mouth pressures, or, better yet, by transdiaphragmatic pressure, to determine whether frequent stretching affects strength.

In conclusion, the report herein by Kakizaki et al presents excellent food for thought and an opportunity to review current concepts of respiratory mechanics and dyspnea. The authors and readers should take up the challenge of testing and advancing this hypothesis in pursuit of more effective treatment of dyspnea and improving the functional capacity and quality of life of dyspnea patients.

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The views expressed herein reflect only the views of the author and are not the official views of the Department of the Army or the Department of Defense.

Preliminary Report on the Effects of Respiratory Muscle Stretch Gymnastics on Chest Wall Mobility in Patients With Chronic Obstructive Pulmonary Disease

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OBJECTIVE: To examine the effect of respiratory muscle stretch gymnastics (RMSG) on chest wall mobility, pulmonary function, and dyspnea in daily living in patients with chronic obstructive pulmonary disease (COPD). PATIENTS AND METHOD: The subjects were 22 consecutive COPD patients who were regularly treated in the outpatient clinic of a medical university rehabilitation hospital. The patients did not have severe limitations in the range of movement in the shoulders, and were unfamiliar with RMSG. Chest wall mobility (difference between chest circumference during deep expiration and deep inspiration), pulmonary function test (forced expiratory volume in 1 s [FEV_1] and vital capacity), and dyspnea in daily living (Fletcher's rating) were measured before and after 4 weeks of RMSG. Four RMSG patterns were demonstrated to each patient to ensure that they could perform the gymnastics without assistance. The patients were instructed to perform each pattern four times during each session (3 sessions per day) for 4 weeks, at which time, the patients were asked to return for re-evaluation. RESULTS: Chest wall expansion and reduction increased at both the upper (0.8 ±0.2 and 1.3 ± 0.2 cm, respectively) and lower (0.4 ± 0.2 and 0.7 ± 0.2 cm, respectively) chest walls. Vital capacity increased 119 ± 43 mL, while FEV_1 remained unchanged. Fletcher's rating improved in 12 patients and remained unchanged in 10; it did not worsen in any of the 22 patients. CONCLUSION: RMSG increases chest wall mobility, possibly by reducing chest wall elastance in patients with COPD. [Respir Care 1999:44(4):409–414] *Key words: Chronic obstructive pulmonary disease, COPD, respiratory muscles, pulmonary function, pulmonary rehabilitation, dyspnea.*

Background

At present, respiratory rehabilitation programs may include education, lower and upper extremity exercise training, and psychosocial and behavioral components, including treatment of anxiety or depression, smoking cessation, nutrition intervention, and other modalities.[1] The goals of rehabilitation include relief of dyspnea, improved health-related quality of life, and increased functional capacity.

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However, respiratory rehabilitation of patients with chronic obstructive pulmonary disease (COPD) is evolving, partly because the pathology of COPD, especially the dyspnea, is not fully understood. Afferent activity from the chest wall

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respiratory muscles may play a role in modifying the dyspneic sensation.[2–4] A stiff chest wall or one with increased elastance,[5] which is often observed in clinical practice, may elicit an increase in muscle spindle firing from the noncontracting intercostal muscles[6] and could be responsible, in part, for the dyspneic sensation.[7]

A new component of respiratory rehabilitation programs, called respiratory muscle stretch gymnastics (RMSG),[7–9] has been designed to stretch the respiratory muscles, mainly the chest wall muscles, and to decrease chest wall elastance. An immediate increase in forced vital capacity (VC) (from 1807 ± 141 to 1923 ± 145 mL) in 34 patients[7] and a decrease in functional residual capacity (from 4.19 ± 1.27 to 3.88 ± 1.03 L) in 12 patients after 4 weeks[9] have been reported as a result of RMSG. Other clinical benefits of short-term[7] and long-term[9] RMSG include decreased dyspnea at rest and after a 6-min walking test, improved quality of life, prolonged 6-min walking distance, and decreased residual volume, total lung capacity, and residual volume/total lung capacity.

It has been suggested that reduced chest wall mobility is associated with a decrease in lung function and exercise endurance.[10] It is possible that RMSG increases VC and exercise endurance by reducing chest wall elastance, causing increased chest wall mobility. Chest wall expansion and reduction have been used by physical therapists to measure chest wall mobility.[11] Herein, we report our preliminary clinical experience with RMSG from a physical therapist's point of view, with particular reference to the effect on chest wall mobility, dyspnea, and spirometry.

Methods

Subjects

The subjects (Table 1) were 22 consecutive patients (19 men, 3 women; mean age, 73.4 ± 1.0 years [range, 62–81]) with COPD but without severe limitations in range of movement in the shoulders. The subjects were regularly treated as outpatients at Showa University Fujigaoka Rehabilitation Hospital. COPD was diagnosed and rehabilitation was indicated according to standard criteria.[12] All patients were in stable condition while receiving standard medical treatment prior to RMSG. Theophylline was used in 20, a β-2 stimulant was used in 11, an anticholinergic agent was used in 12, and steroid was used in 8 patients. There was no change in medication during the observation period. The patients did not have any other significant disease, such as unstable cardiac disease or acute infectious disease; all patients willfully participated in the rehabilitation program and gave informed consent. The patients were unfamiliar with the effect of RMSG on dyspnea. The protocol was approved by the Showa University Ethics Committee.

Measurements

Chest wall expansion and reduction were measured according to the standard method.[11] In brief, a flat tape was placed around the patient's chest, and with arms down, the patient was asked to breathe out as much as possible while the measuring tape was drawn taut and chest circumference was measured (E1); then the tape was released, and the patient was asked to breathe in as deeply as possible for the next measurement (I; ie, deep inspiration). The patient was again asked to breathe out as far as possible for the final measurement (E2). Measurements were done at the axillary level (upper chest wall) and the xiphisternum level (lower chest wall). Each measurement was repeated twice, and the average value was used. All patients also underwent spirometry before and after 4 weeks of RMSG. Testing and quality control followed standard and recommended procedures.[13]

Protocol

Chest wall mobility was measured before RMSG. Spirometric values obtained when the patients were in a stable condition during routine clinical follow-up within a few months before RMSG were used as the pre-RMSG values. Dyspnea was rated according to Fletcher's classification[14] before RMSG. The 4 RMSG patterns[7] were demonstrated to each patient to ensure that they could perform the gymnastics without assistance. The exercises, described below, were each done 4 times in a specified order. Patients were instructed to do 3 sessions of exercises per day—one in the morning, afternoon, and evening—for 4 weeks. After 4 weeks of RMSG, an independent evaluator observed each patient to determine their ability to perform RMSG. The results of this evaluation were not revealed until all other measurements and ratings (ie, chest wall mobility, spirometry, and dyspnea) were completed. We inquired as to the patient's compliance to perform RMSG but did not ask for quantitative ratings. All patients stated that they had high compliance.

Respiratory Muscle Stretch Gymnastics

Pattern 1. Elevating and Pulling Back the Shoulders and Stretching the Upper Chest. Slowly breathe in through the nose while gradually elevating and pulling back both shoulders. After taking a deep breath, slowly breathe out through the mouth, lower the shoulders, and relax.

Pattern 2. Pulling Down and Stretching the Upper Chest. Place both hands on the upper chest. Pull back elbows and pull down chest while lifting the chin and inhaling deeply through the nose. Expire slowly through the mouth and relax.

Table 1. Patient Profiles for 22 Patients with Chronic Obstructive Pulmonary Disease

Patient No.	Age (years)	Sex	Height (cm)	Weight (kg)	VC (L)	FEV_1 (L)	FEV_1%	Fletcher's Rating	
								Before RMSG	After RMSG
1	79	M	163	50	3.48	2.53	69.9	4	3
2	73	M	153	41	2.15	0.58	26.9	4	3
3	71	M	167	43	2.13	0.84	42	3	3
4	70	M	161	47	1.8	0.72	40	3	2
5	78	M	158	55	2.61	1.1	43.3	4	3
6	81	M	174	48	2.19	0.73	34.8	4	3
7	73	F	150	60	1.75	0.92	53.5	3	3
8	78	M	175	58	2.46	1.83	74.1	3	2
9	71	M	164	48	3.07	1.07	33.3	3	3
10	69	M	150	38	1.97	0.47	25.8	4	3
11	78	F	142	42	1.81	0.52	30.1	3	2
12	78	F	156	57	1.99	0.97	47.8	3	3
13	70	M	165	53	2.87	1.93	69.9	3	3
14	67	M	169	65	3.5	1.02	29.8	3	2
15	76	M	160	50	2.28	0.92	45.09	3	3
16	78	M	159	50	1.7	0.69	43.1	3	2
17	67	M	167	74	1.99	0.74	38.7	2	2
18	77	M	165	50	2.89	1.15	39.9	3	2
19	62	M	166	70	3.73	1.35	38.7	2	2
20	73	M	161	47	3.13	0.88	28.7	3	3
21	73	M	173	53	3.3	1.21	38.3	2	2
22	72	M	157	38	2.54	0.88	45.83	3	2
Mean	73.36		161.59	51.68	2.52	1.05	42.71		
SE	1.02		1.76	2.03	0.14	0.11	2.90		

VC = vital capacity; FEV_1 = forced expiratory volume in 1 s; RMSG = respiratory muscle stretch gymnastics.

Pattern 3. Arching and Stretching the Back. Hold hands in front of chest. While slowly breathing in through the nose, arch upper back, moving hands to front and down, and stretch the back. After deep inspiration, slowly breathe out and resume the original position.

Pattern 4. Raising the Arms and Stretching the Lower Chest. Hold the ends of a face towel with both hands stretched out at shoulder height. After taking a deep breath, move arms up while breathing out slowly. After deep expiration, lower hands and breathe normally.

Pattern 5. Elevating the Elbow and Stretching the Lower Chest. Hold one hand behind the head and pull the elbow to the side. Breathe deeply through the nose, and while slowly exhaling through the mouth, stretch the trunk of the body by raising the elbow as high as is easily possible. Return to original position while breathing normally. Repeat the process, using the alternate hand behind the head.

Statistical Data

All data are shown as the average ± SE. Statistical comparisons between measurements before and after RMSG were calculated using paired Student's *t* test, unless otherwise specified.

Results

Chest wall expansion (from 3.4 ± 0.3 to 4.2 ± 0.3 cm; $p < 0.01$) and reduction (from 2.9 ± 0.3 to 4.1 ± 0.3 cm; $p < 0.0001$) in the upper chest wall significantly increased after 4 weeks of RMSG (Fig. 1). This could be due to the increase in the deep inspiration measurement after RMSG compared with the baseline value (from 87.3 ± 1.0 to 88.1 ± 0.9 cm; $p < 0.01$), whereas the E1 (from 83.9 ± 1.0 to 83.9 ± 1.0 cm) and E2 (from 84.4 ± 1.1 to 83.9 ± 1.0 cm) measurements remained unchanged. Before RMSG, the chest expansion was significantly larger than the reduction ($p < 0.001$), which could be due to the E2 value (84.4 ± 1.1 cm) being significantly ($p < 0.001$) larger than the E1 value (83.9 ± 1.0 cm), but this difference was not seen after RMSG.

Chest wall expansion (from 4.9 ± 0.4 to 5.3 ± 0.4 cm; $p < 0.05$) and reduction (from 4.5 ± 0.4 to 5.2 ± 0.4 cm; $p < 0.001$) in the lower chest wall also significantly increased after 4 weeks of RMSG (Fig. 2). The increase in chest wall reduction could be due to the decrease in the E2

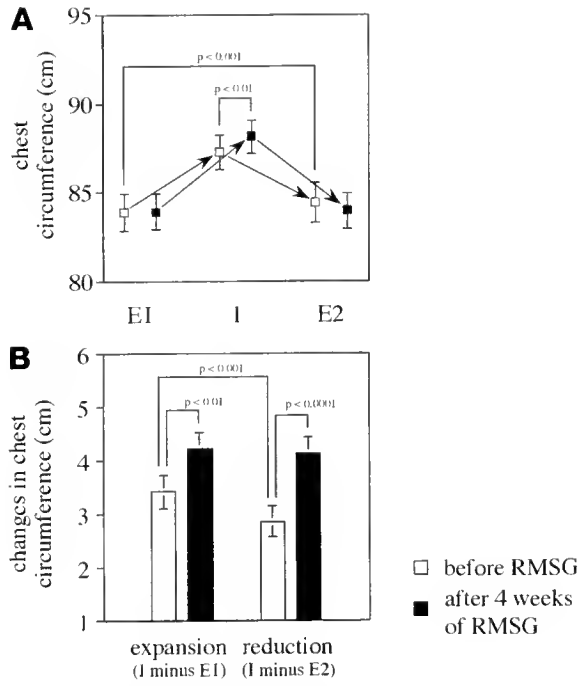

Fig. 1. A, mean ± SE of upper chest circumference during deep expiration (E1), deep inspiration (I), and subsequent expiration (E2) before and after respiratory muscle stretch gymnastics (RMSG) in 22 patients with chronic obstructive pulmonary disease. B, upper chest expansion and reduction.

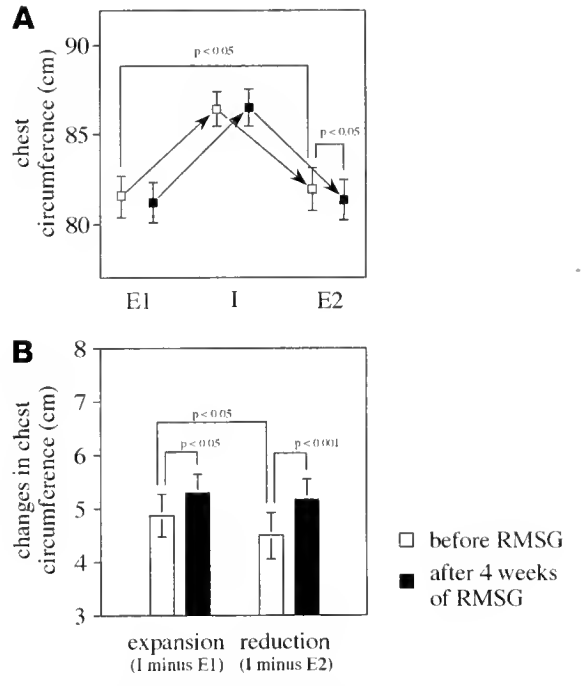

Fig. 2. A, mean ± SE of lower chest circumference during deep expiration (E1), deep inspiration (I), and subsequent expiration (E2) before and after respiratory muscle stretch gymnastics (RMSG) in 22 patients with chronic obstructive pulmonary disease. B, lower chest expansion and reduction.

measurement after RMSG compared with the baseline value (from 81.9 ± 1.2 to 81.3 ± 1.1 cm; $p < 0.05$). Before RMSG, chest expansion was significantly larger than chest reduction ($p < 0.05$), which could be due to the E2 value (81.9 ± 1.2 cm) being significantly ($p < 0.001$) larger than the E1 value (81.5 ± 1.2 cm), but the difference disappeared after RMSG.

Table 1 shows Fletcher's rating of dyspnea before and after 4 weeks of RMSG. The rating improved in 12 patients and remained unchanged in 10; it did not worsen in any of the 22 patients. The overall improvement was statistically significant (sign test, $p < 0.005$). The pre- and post-RMSG ratings and change in ratings did not correlate with either the measurement of the chest circumference or spirometric values. However, there was a negative linear correlation between the pre-RMSG ratings and the change in rating (Pearson's correlation coefficient = −0.60; $p < 0.005$), suggesting that those with more severe symptoms had a greater likelihood of benefiting from RMSG. All 5 patients who had a baseline Fletcher's rating of 4 improved to 3 after 4 weeks of RMSG.

Figure 3 compares patients who were considered to have good ($n = 10$) and poor ($n = 12$) RMSG performance on chest expansion at the upper chest wall level. Before RMSG, there was no difference in chest expansion between the 2 patient groups (3.7 ± 0.5 vs 3.2 ± 0.4 cm; $p = 0.42$). Both groups had significantly increased chest expansion ($p < 0.05$). However, after 4 weeks of RMSG, chest expansion in the patients with good performance (4.9 ± 0.4 cm) was significantly ($p < 0.05$) greater than that in the patients with poor performance (3.7 ± 0.4 cm). Similarly, the reduction in upper chest wall before RMSG did not differ between the 2 groups, but after RMSG, the patients with good performance had a larger upper chest wall reduction than those with poor performance. The 2 groups did not differ in expansion or reduction of the lower chest wall before and after RMSG.

VC significantly increased 119 ± 43 mL (from 2.515 ± 0.136 to 2.634 ± 0.139 L; $p < 0.05$) after 4 weeks of RMSG (Fig. 4). Forced expiratory volume in 1 second (FEV_1) was 1.05 ± 0.11 L before RMSG and 1.08 ± 0.10 L after RMSG, which was not a statistically significant difference.

Discussion

The results indicate that RMSG increases chest wall mobility in patients with COPD. Because this is a report on our clinical observation before and after RMSG without a control group and without bias elimination, the results may not be conclusive; however, chest wall compliance is known to decrease with age[15] and with COPD.[5]

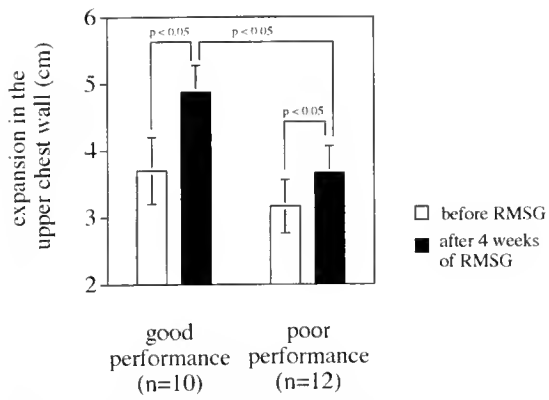

Fig. 3. Mean ± SE of the expansion in upper chest wall before and after 4 weeks of respiratory muscle stretch gymnastics (RMSG) in patients with good and patients with poor RMSG performance.

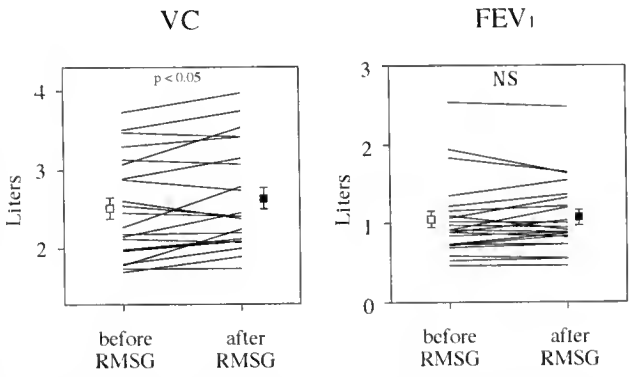

Fig. 4. Vital capacity (VC) and forced expiratory volume in 1 s (FEV_1) before and after 4 weeks of respiratory muscle stretch gymnastics (RMSG) in 22 patients with chronic obstructive pulmonary disease. Lines indicate Individual results; boxes indicate group mean ± SE. NS = not significant.

Also, forced VC is reported to decrease with age.[16] Together, these findings may indicate that chest wall mobility decreases with time. It has been reported that decreased chest wall mobility is associated with low pulmonary function and exercise intolerance.[10] In the present report, chest wall mobility was significantly increased after 4 weeks of RMSG. It is very unlikely that this increase occurs simply with time or because the patient's disease state has rapidly improved.

Thus, it may be reasonable to suggest that RMSG has the potential to increase chest wall mobility in patients with COPD. This is further supported by the results that after 4 weeks of RMSG, upper chest wall mobility in patients with good RMSG performance was greater than that in the patients with poor RMSG performance. Since the baseline values, including the variables listed in Table 1, did not vary significantly between the 2 groups, the difference may indicate the effect of RMSG to increase chest wall mobility. It has been shown that stretching exercises increase the range of motion in the extremities.[17] Thus, it may be reasonable to suggest that RMSG can also increase the range of motion of the chest wall.[7,9] Some of the improvements in expansion and VC did reach statistical difference, but the difference was small in magnitude. However, overall improvements in reduction were larger, namely 1.3 and 0.7 cm in the upper and lower chest wall, respectively.

The increase in chest wall mobility after RMSG could be due to reduced elastance (ie, pressure change over volume change) in the chest wall skeletal system or to an increase in respiratory muscle force (or both). The present report and others[7] suggest that RMSG increases VC in COPD patients. Since decreased VC is believed to indicate restriction of the respiratory system, RMSG might have the potential of decreasing the elastance of the respiratory system. RMSG had no effect on FEV_1, which makes it unlikely that it had an effect on the lungs or airways. Thus, the RMSG-induced decrease in the elastance of the entire respiratory system could be due to a decrease in chest wall elastance. It is also possible that RMSG increased the respiratory muscle force and, thus, increased chest wall mobility. However, from the nature of the gymnastic, this is unlikely to be the case. As previously reported, the maximum inspiratory pressure after 4 weeks of RMSG did not change from baseline.[18]

In our investigation and others,[7,9] dyspnea decreased after 4 weeks of RMSG. We believe that the increase in chest wall mobility and the decrease in dyspnea may be related. The level of central respiratory motor command has been suggested to be associated with dyspnea.[19] The reduction in dyspnea could be due to an increase in chest wall mobility, which leads to a reduction in central motor command for a given level of ventilation. Decreased dyspnea could also be due to altered afferent activity from the intercostal muscle spindles. It has been suggested that increased muscle spindle firing from the noncontracting intercostal muscles elicits dyspnea.[2,4] Muscle spindle firing from the noncontracting muscles is decreased after stretching and after isometric voluntary contraction.[6,20,21] Such a decrease could take place in the intercostal muscles after RMSG and could play a role in the decrease in dyspnea. A large-scale, randomized, controlled study and attempts to measure chest wall elastance are needed.

Conclusion

In this preliminary report, RMSG increased chest wall mobility and VC and decreased dyspnea, possibly by reducing chest wall elastance. Thus, RMSG may provide clinically significant benefits for patients with COPD.

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Long-Term Tracheostomy in Severe COPD Patients Weaned from Mechanical Ventilation

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BACKGROUND: Chronic obstructive pulmonary disease (COPD) patients who suffer from acute respiratory failure (ARF) requiring mechanical ventilation are at risk of relapse. It is unknown whether spontaneously breathing patients benefit from retaining a tracheostomy after discharge from the intensive care unit. We studied the effects of long-term (6 months) tracheostomy in severe COPD patients weaned from mechanical ventilation. METHODS: Twenty tracheostomized COPD patients recovering from ARF and weaned from mechanical ventilation were randomly allocated into 2 groups: 10 patients were maintained on tracheal cannula; 10 patients had the tracheal cannula removed (cutaneous fistula spontaneously closed). Breathing pattern, forced lung volumes, respiratory muscle force, and arterial blood gases were evaluated at discharge and at 1, 3, and 6 months after discharge. Hospitalized days, mortality rate, and number of new exacerbations requiring antibiotics were recorded. Maximal expiratory pressure (but not other lung function parameters) significantly improved in both groups. In both groups, 2 out of 10 patients died due to respiratory causes after 5.0 ± 0.8 months after discharge. During the follow-up period, exacerbations were significantly greater in the tracheostomized patients than in those whose tracheostomies had been removed before discharge, though there was no significant difference in hospitalized days between the 2 groups. CONCLUSION: Chronic tracheostomy in severe COPD patients is associated with a higher frequency of exacerbations requiring antibiotic treatment. Unless there are absolute indications for tracheostomy, COPD patients weaned from mechanical ventilation should undergo early decannulation. [Respir Care 1999;44(4):415–420] *Key words: respiratory failure, chronic obstructive pulmonary disease, mechanical ventilation, airway infection, weaning, decannulation.*

Introduction

Prolonged mechanical ventilation is usually delivered through a tracheostomy.[1] Although an artificial airway is often maintained in place because of swallowing dysfunction, inability to clear secretions, or continuous need for mechanical ventilation,[1,2] the appropriate time for tracheostomy removal after weaning and in the absence of the latter conditions is debated.[3] Tracheostomized patients can suffer from chronic infections and increased secretions such that they frequently need suction. Patients on long-term ventilation can be at increased risk of infection,[4] and tracheostomy-related inadequate airway humidification[5] may adversely affect cough reflex and mucociliary clearance. This problem is even more complex in patients with chronic obstructive pulmonary disease (COPD), and patients suffering acute respiratory failure (ARF) requiring mechanical ventilation are at risk of relapse.[6,7] Therefore, despite the aforementioned potential complications of long-term tracheostomy, it is conceivable that maintaining a tracheostomy in spontaneously breathing patients after discharge from the intensive care unit (ICU) can avoid repeated tracheostomies and perhaps improve convalescence.

We conducted a prospective, randomized, controlled study to evaluate the effect (on both course and outcome) of maintaining a tracheal cannula in discharged spontaneously breathing severe COPD patients.

Methods

The study was approved by the Ethical Committee of the Medical Center of Gussago, S Maugeri Foundation

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Table 1. Demographic, Anthropometric and Last Stable Functional Characteristics of Patient Population

Parameter	Group 1	Group 2	p-value
Age, years	71 ± 6	67 ± 8	ns
Sex, M/F	8/2	8/2	ns
BMI	25 ± 6	22 ± 4	ns
FEV_1, mL	737 ± 210	557 ± 225	ns
FEV_1, %pred	30 ± 10	24 ± 15	ns
FVC, mL	1271 ± 413	1154 ± 415	ns
FVC, %pred	40 ± 11	41 ± 19	ns
RV, %pred	153 ± 27	155 ± 30	ns
TLC, %pred	144 ± 31	146 ± 36	ns
MIP, %pred	38 ± 7	36 ± 6	ns
MEP, %pred	50 ± 23	61 ± 13	ns
P_{aCO_2}, (mm Hg) (on oxygen)	51 ± 6	57 ± 9	ns
P_{aO_2}, (mm Hg] (on oxygen)	70 ± 7	76 ± 12	ns
P_{aO_2}/F_{IO_2} (mm Hg)	2.54 ± 0.27	2.53 ± 0.40	ns
LTOT, %	40	70	<0.01
Previous ARF, no./year*	0.5 ± 0.7	0.8 ± 1.1	ns

Group 1: cannula retained. Group 2: cannula removed. BMI = body mass index; FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity; RV = residual volume; TLC = total lung capacity; MIP = maximal inspiratory pressure; MEP = maximal expiratory pressure; P_{aCO_2} = arterial carbon dioxide tension; P_{aO_2} = arterial oxygen tension; F_{IO_2} = fraction of inspired oxygen; LTOT = long term oxygen therapy; ARF = acute respiratory failure; %pred = percent of predicted value. *Previous ARF data calculated on the 2 years preceding the study.

IRCCS, and was conducted according to the Declaration of Helsinki. Patients gave their informed consent to participate in the study.

Patient Population

We studied 20 patients with severe COPD (as defined by the American Thoracic Society guidelines[8]) who were recovering from ARF and admitted to our respiratory intermediate ICU (RIICU) to complete the weaning process from prolonged mechanical ventilation. Demographic, anthropometric, and functional characteristics of the patients when in their last stable condition are shown in Table 1. All of the patients were severely obstructed and hyperinflated. Their regular medical treatment consisted of inhaled bronchodilators. Eleven patients were on long-term oxygen therapy, none were on domiciliary mechanical ventilation, but 12 of the 20 patients had experienced noninvasive positive pressure ventilation for acute exacerbations of their disease before the study. All of them had been transferred from ICUs of other hospitals, had undergone a percutaneous tracheostomy after 15 ± 3 days, and invasive mechanical ventilation was prolonged for 25 ± 5 days. Causes of ARF were exacerbation[9] and pneumonia in 54% and 46% of cases respectively. At admission, patients needed ventilatory assistance for 14 ± 3 hours per day. Successful weaning[10] was obtained 9 ± 5 days after RIICU admission, after which patients breathed through an uncuffed tracheal cannula with an expiratory valve that allowed them to speak and cough efficiently.*

The study inclusion criteria were: (1) unassisted spontaneous breathing for at least 48 consecutive hours, (2) no clinical or laboratory sign of infection (leukocytosis, fever, or radiographically-identifiable pulmonary infiltrates), (3) absence of infection as defined by tracheal bacterial pathogens less than 2×10^5 cfu·mL^{-1} on a culture strain (specimen obtained via fiberoptic tracheobronchoscopy), (4) no current need for antibiotics, and (5) no change in drugs or in oxygen requirement during the last 7 days.

Study exclusion criteria were: (1) conditions indicating an absolute need for maintaining tracheostomy, such as swallowing or vocal cord dysfunction, obstructive tracheal lesions (granuloma or stenosis, as assessed by fiberoptic tracheobronchoscopy), or inability to spontaneously clear secretions, (2) systemic diseases, (3) cancer, and (4) inadequate self-care or familial assistance.

Measurements

Breathing pattern (tidal volume [V_T], respiratory rate [f], and minute ventilation [$\dot{V}_E$]), and forced lung volumes (forced expiratory volume in 1 second [FEV_1] and forced vital capacity [FVC]) were measured with a portable spirometer and compared with the values predicted by Quanjer.[11] The best values of 5 measurements were considered for analysis. Respiratory muscle strength was assessed by measuring maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) at the mouth using a respiratory module system, starting from the level of functional residual capacity (in measuring MIP) and total lung capacity (in measuring MEP), according to the method described by Black and Hyatt.[12] A visual on-line spirographic control during tidal breathing allowed us to control the starting level of the maneuver. MEP and MIP measurements were compared with the predicted values of Bruschi et al.[13] Patients performed consecutive maneuvers (with ≥ 1 minute interval between efforts) until 2 acceptable values differing by ≤ 5% were obtained. The best value was recorded. Spirometry and respiratory muscle function were measured through a mouthpiece while the cannula was closed with a cap. Arterial blood was sampled at the radial artery with the patient in the sitting position and while breathing a fraction of inspired oxygen (F_{IO_2}) that maintained arterial oxygen saturation at ≥ 90% for ≥ 1 hour. Arterial oxygen tension (P_{aO_2}), arterial carbon dioxide tension (P_{aCO_2}), and pH were measured with a Ciba Corning 840 analyzer. P_{aO_2}/F_{IO_2} ratio was measured

* Suppliers of commercial products are identified in the Product Sources section at the end of the text.

while patients were receiving oxygen via nasal prongs, and F_{IO_2} was calculated with the following conversion factor: $F_{IO_2} = 21 + (3 \times$ oxygen flow in L/min of supplemental oxygen).[14]

This procedure was deemed acceptable because the tracheal cannula (with the valve inserted) allowed normal oxygen delivery from the nose through the tracheostomy.

During the follow-up, the cumulative number of days spent in hospital due to respiratory causes, the number of exacerbations requiring antibiotic use, and mortality were recorded from the hospital registers and interviews with the patients' general practitioners. Exacerbation of COPD was defined by worsening of symptoms, fever, increased purulent sputum requiring changes to normal treatment, short courses of antibiotics, oral steroids, and other bronchodilators.[15] Decisions regarding treatment of exacerbation and hospital admission were made by the general practitioner, who was not aware of the purpose of the study.

Microbiology

Cultures were performed on specimens from bronchoscopic protected specimen brush in both groups, processed with the methods described in Bartlett et al.[16] We used N-acetylcysteine to homogenize the sputum specimen.[17] Inoculation of homogenized sample was performed in the following agar media: Trypticase soy + 5% Mutton Blood, Chocolate II + Isovitalex, Mannitol salt, McConkey II, Sabouraud dextrose. All cultures were incubated at 37° C under aerobic and anaerobic conditions and in carbon dioxide-enriched atmosphere. Cultures were evaluated for growth after 24 hours and 48 hours, and, if negative, discarded after 5 days. Bacterial agent colonization was defined as the isolation of a potential pathogen in sputum culture, in the absence of signs or symptoms of lower respiratory tract infection.[18,19] Samples were transferred within 1 hour to the laboratory. All the potential isolated pathogen micro-organisms were identified by standard laboratory methods.[20]

Study Design

After inclusion criteria were verified, patients were randomly assigned to 2 groups: Group 1 (cannula retained) and Group 2 (cannula removed and cutaneous fistula spontaneously closed). The retained cannulae were fitted with a valve that facilitates speaking and coughing. Measurements were performed at the time of randomization (TR), at the time of discharge (T0), and during the follow-up at 1, 3, and 6 months after discharge (T1, T3, and T6, respectively).

Statistics

Results are shown as mean ± SD. Frequencies were used to describe the microbiological variables and the number of exacerbations requiring use of antibiotics. Mortality was assessed by percentage rate. Analysis of variance was used to test differences between and within groups. Whenever necessary, a post hoc test with Bonferroni correction was used to evaluate the contrast between and within groups. Differences in frequency distribution of exacerbations and microbiology before randomization were tested by means of an additional Fisher's exact test. A p value < 0.05 was considered statistically significant.

Results

Demographic, anthropometric and functional characteristics of patients when in their last stable condition are shown in Table 1. The groups differed only in the use of long-term oxygen therapy, which was more frequent in the patients of Group 2. Mean duration of RHCU stay was not different between the groups (Group 1: 16 ± 8 days; Group 2: 19 ± 10 days).

The course of breathing patterns, forced lung volumes, respiratory muscle strength, and arterial blood gases are shown in Table 2. No significant differences were observed between the groups at any time. Only MEP significantly improved over time in both groups ($p < 0.005$). Oxygen requirement over time did not change in either group: P_{aO_2}/F_{IO_2} ratio also remained unchanged (see Table 2).

Table 3 shows the bacteria species present in the airways before randomization. No significant differences were seen between the groups. The total bacterial growth was similar between groups, the value ranging from 2×10^2 to 2×10^5 cfu·mL^{-1}.

One subject of Group 2 had to be restarted on mechanical ventilation with a new tracheostomy because of severe exacerbation 18 days after discharge. None of the patients died in hospital. Two out of 10 patients (20%) in both groups died after a mean of 5.0 ± 0.8 months after discharge due to ARF requiring ICU admission. The causes of ARF were pneumonia (2 patients from Group 1 and 1 patient from Group 2) and severe exacerbation (1 patient from Group 2). One patient from Group 1 underwent accidental self-decannulation during the follow-up at the 5th month; the cannula was not replaced and the fistula closed spontaneously. At the end of the follow-up all the surviving patients from Group 1 were decannulated.

The cumulative number of days spent in hospital during the follow-up was not significantly different (Group 1: 10.2 ± 9.7 days per patient; Group 2: 4.5 ± 2.7 days per patient). The frequency distribution of exacerbations requiring use of antibiotics is shown in Figure 1. All the patients from Group 1 suffered at least 1 exacerbation during the follow-up, whereas only 5 of the 10 patients in Group 2 required antibiotic therapy for exacerbations ($p < 0.005$).

Table 2. Time Course of Respiratory Functional Parameters

Variable	Group	TR (G1 = 10, G2 = 10)	T0 (G1 = 10, G2 = 10)	T1 (G1 = 10, G2 = 9)	T3 (G1 = 10, G2 = 9)	T6 (G1 = 10, G2 = 9)	p-value
V_T (mL)	1	509 ± 122			470 ± 69	466 ± 64	ns
	2	434 ± 73			445 ± 42	450 ± 53	ns
V_E (L/min)	1	8.6 ± 2.8			7.1 ± 1.0	7.1 ± 1.2	ns
	2	6.8 ± 1.7			6.5 ± 0.7	6.8 ± 0.5	ns
FEV_1 (%pred)	1	30 ± 10			31 ± 8	33 ± 8	ns
	2	24 ± 15			22 ± 16	25 ± 9	ns
FVC (%pred)	1	40 ± 11			42 ± 10	43 ± 8	ns
	2	41 ± 19			44 ± 12	43 ± 7	ns
MIP (%pred)	1	38 ± 7	40 ± 8	41 ± 11	43 ± 10	43 ± 15	ns
	2	36 ± 6	37 ± 7	42 ± 11	39 ± 8	41 ± 8	ns
MEP (%pred)	1	50 ± 23	51 ± 20	61 ± 26	60 ± 24	62 ± 22	< 0.005
	2	60 ± 13	56 ± 17	73 ± 8	68 ± 20	72 ± 14	< 0.005
P_{aO_2}/F_{IO_2} (mm Hg)	1	2.60 ± 0.53	2.67 ± 0.71	2.58 ± 0.54	2.51 ± 0.38	2.53 ± 0.39	ns
	2	2.75 ± 0.55	2.81 ± 0.38	2.66 ± 0.65	2.70 ± 0.44	2.76 ± 0.34	ns
P_{aCO_2} (mm Hg)*	1	51 ± 6	46 ± 3	46 ± 6	49 ± 6	48 ± 5	ns
	2	59 ± 9	55 ± 12	55 ± 8	52 ± 9	54 ± 12	ns

TR = measurements at time of randomization; T0 = measurements at time of discharge; T1, T3, and T6 = measurements at 1 month, 3 months, and 6 months, respectively; G1 = Group 1; G2 = Group 2; V_T = tidal volume; ns = not significant; $\dot{V}_E$ = minute ventilation; FEV_1 = forced expiratory volume in one second; %pred = percent of predicted value; FVC = forced vital capacity; MIP = maximal inspiratory pressure; MEP = maximal expiratory pressure; P_{aO_2} = arterial oxygen tension; F_{IO_2} = fraction of inspired oxygen; P_{aCO_2} = arterial carbon dioxide tension. * = on oxygen.

Table 3. Microbiology Data at Time of Randomization

	Ps. aeruginosa	S. aureus	Acinetobacter spp.	K. pneumoniae	Enterobacter spp.	Morganella spp.
Group 1 (n = 10)	6	4		2		1
Group 2 (n = 10)	8	2	1	2	1	

Numeric data show numbers of patients with sputum positive for specific infectious agent. Each patient may be counted one or more times into the different columns

Discussion

Our study shows that in severe COPD patients weaned from mechanical ventilation after an episode of ARF, chronic tracheostomy does not improve short-term survival or lung function, and that chronic tracheostomy is associated with a higher rate of exacerbations requiring antibiotics.

Mechanical ventilation via tracheostomy is a well recognized modality in the management of ARF. In patients who are slow to recover, the artificial airway often remains in place because of the patient's inability to clear secretions, swallowing dysfunction, or the need for prolonged mechanical ventilation.[1,2] However, chronically tracheostomized patients can suffer from several problems, including inadequate humidification (due to poor hygiene, high flow of cool oxygen, risk of exposure in a cool climate),[5,21] tracheal lesions,[4,22] higher incidence of bacterial colonization of the distal airways,[18,19] and increased risk of infection when associated with prolonged mechanical ventilation.[23,24]

In clinical practice—in patients weaned from mechanical ventilation—tracheostomy removal is often delayed after discharge, on the reasoning that prolonging tracheostomy might help the most severe and functionally compromised patients reach a better clinical condition before removing the cannula. Survival at 1 year in COPD patients undergoing endotracheal intubation and mechanical ventilation is variable but low.[10] Vitacca et al[25] found that severe COPD patients requiring invasive mechanical ventilation after an episode of ARF show a poor prognosis within the following year: mortality rate was 26% in the ICU, 48% at 3 months, and 63% at 1 year. In that study the number of new ICU admissions during 1-year follow-up was 0.12 versus 0.30 per patient. In our study 6-month mortality rate was 20%, similar to that reported by Nava et al[10] in COPD patients successfully weaned from prolonged (more than 21 days) mechanical ventilation via tracheostomy. Diaphragmatic fatigue, high airway resistance, lung hyperinflation, hypoxemia, hypercapnia, malnutrition, and recurrent infections can all affect long-term survival.[6,7] Our results might indicate that tracheostomy per se does not adversely affect the prognosis of COPD patients successfully weaned from mechanical ventilation.

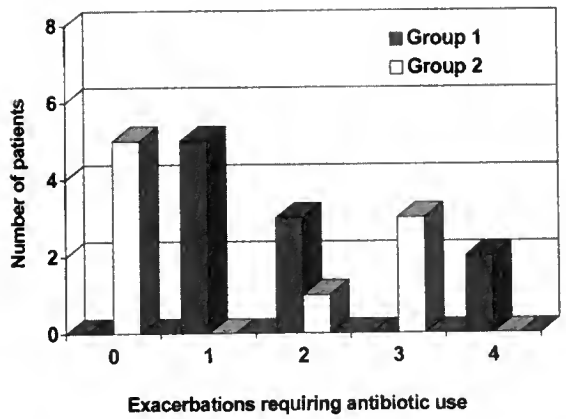

Fig. 1. Exacerbations during the 6 months of follow-up. Y-axis: cumulative number of patients suffering exacerbation. X-axis: frequency distribution of exacerbations requiring antibiotics. p value < 0.005 for differences tested by Fisher's χ^2 analysis.

All the patients showed a severe impairment in respiratory function, with no differences between the 2 groups despite a slight but not significant difference in FEV_1/FVC ratio mostly due to the small sample size. Follow-up measurements were taken differently in the 2 groups because of the presence of the tracheostomy in Group 1. However, there was no difference in spirometric function between the 2 groups, taken at discharge after randomization (partial available data not presented). Most of the patients (60%, equally distributed in the 2 groups) had experienced episodes of ARF treated with noninvasive mechanical ventilation during the 2 years prior to the study (see Table 1). The only significant difference between the 2 groups was the previous use of long-term oxygen therapy, which was higher in Group 2 (tracheostomy removed). This might constitute a bias that favored Group 1 (tracheostomy retained).

Patients of Group 1 were discharged with an uncuffed tracheal cannula with an expiratory valve, but without a humidification system. In a previous study,[21] we found significant short-term improvements in respiratory function and in tracheal secretions over 10 days by applying a hygroscopic condenser humidifier. It is unknown whether the use of a humidifier might have resulted in improved respiratory function in Group 1. Both groups showed a significant improvement in MEP. Whereas MIP depends mainly on lung hyperinflation and mechanics,[26] MEP better reflects the patient's fitness and is directly related to the improvement of peak cough flows and spontaneous cough reflex.[27] From a pathophysiological point of view, retaining a tracheostomy might reduce the dead space ventilation and the work of breathing, though these 2 aspects were not assessed in the present study.

We considered whether the presence of a tracheostomy valve in Group 1 created a favourable bias, and concluded that the bacterial exacerbation results do not support that hypothesis. Before entering the randomization, the number of patients with sputum positive for potential infectious agents was not significantly different between the groups (see Table 3). Airway bacterial colonization is common in stable patients with chronic lung disease, as well as in patients with long-term tracheostomy.[18,19] Bacterial distribution in our sample of patients showed a prevalence of Gram-negative strains, as has been reported in chronically ill patients.[23] In contrast to the report of de Latorre et al,[24] we observed a significant prevalence of *Pseudomonas aeruginosa*. *Pseudomonas aeruginosa* and *Staphylococcus aureus* are well-known contaminants of the hospital environment, and colonizing species during mechanical ventilation in the ICU.[24] Contamination could easily have been introduced by routine suctioning or humidification, ambient air during the weaning trial, a nasal sinus infection, the ventilator circuit, or the tracheostomy cuff.

In 49 sets of cultures on 15 subjects with long-term tracheostomy, Niederman et al[23] found that patients with persistent tracheobronchial colonization were more ill and developed tracheobronchitis more often than those without persistent tracheobronchial colonization. A limitation of our study is that there was no microbiological evaluation during the follow-up period. This aspect of data collection was not possible in this case for technical reasons—the same bronchoscopic technique used to sample bronchial secretions could not be easily applied in the ambulatory setting, and sputum sample is not as reliable a means of sample collection as bronchial brush or bronchoalveolar lavage, a more appropriate means to identify pathogens.[28] Thus, there was no way to correlate bronchial microbiological data and clinical outcome in terms of new exacerbations during the follow-up. In our study, all the patients from Group 1 experienced at least one exacerbation requiring antibiotic use, but only 4 patients from Group 2 experienced an exacerbation requiring antibiotic use. This suggests that prolonged tracheostomy plays an adverse role in the management of COPD patients. However, the lack of standardization of antibiotic use and the small population of this study should make us very cautious in interpreting this interesting result. Moreover, the causes of the increased tracheobronchial infection rate in Group 1 are unknown. Coughing is an important aspect of effective mucus clearance,[29] and decannulation could have favored a more effective cough reflex and thereby reduced the probability of exacerbation in Group 2. Furthermore, decannulation could also have reduced the risk of aspiration and related exacerbation or pneumonia.[30]

Conclusion

Although the number of days spent in hospital due to respiratory causes was not different between the 2 groups, our study suggests that chronic tracheostomy might ad-

versely affect the course of severe COPD patients weaned from mechanical ventilation, by increasing the risk of exacerbation. However, long-term tracheostomy does not appear to affect short-term survival in severe COPD patients. While the statistical power of the present study was limited by the small patient population, our results suggest that COPD patients weaned from mechanical ventilation should undergo early decannulation unless there are absolute indications for retaining the tracheostomy.

PRODUCT SOURCES

Cannula:
Biesalsky-type cannula, Biesalsky, Rusch, Germany

Portable Spirometer:
Pony class 1 Type B, Cosmed, Rome, Italy

Respiratory Module System:
RPM System, Medical Graphic Corp, St Paul MN

Blood Gas Analyzer:
Ciba Corning 840, Ciba Corning, Medfield MA

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Work of Breathing during Weaning from Ventilation: Does Extending Weaning with Continuous Positive Airway Pressure Confer Any Advantage?

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BACKGROUND: The advantage of using continuous positive airway pressure (CPAP) during weaning from mechanical ventilation is unproven. METHODS: Forty-two patients were weaned with pressure support ventilation (PSV) following prolonged mechanical ventilation. The 20 patients in Group A were removed directly from PSV after at least 24 hours of PSV_{5+5} (5 cm H_2O of PSV plus 5 cm H_2O of positive end-expiratory pressure). The 22 patients in Group B were weaned for an additional 24 hours on CPAP. Weaning outcome was compared between the 2 groups by χ^2. RESULTS: The work of breathing was lower with PSV_{5+5} versus $CPAP_5$ (5 cm H_2O of CPAP) when all 42 patients were taken together, in the 36 patients who succeeded weaning, and in 6 patients who failed. The work of breathing was not different between Groups A and B for either ventilatory mode. There were no statistical differences between the failure rates in the 2 modes. CONCLUSION: The work of breathing during PSV_{5+5} was lower than during $CPAP_5$. The work of breathing and the weaning outcome were the same in PSV_{5+5} and $CPAP_5$. Extending weaning with $CPAP_5$ following PSV_{5+5} offered no benefit, but instead imposed an additional load on the ventilatory musculature. [Respir Care 1999;44(4):421–427] *Key words: work of breathing, pressure support ventilation, continuous positive airway pressure, bi-level airway pressure, weaning, mechanical ventilation.*

Introduction

Gradual weaning with pressure support ventilation (PSV) improves respiratory muscle strength and endurance in mechanically ventilated patients recovering from prolonged acute respiratory failure.[1,2] This weaning consists of gradually lowering the inspiratory pressure to a low level,[2] optionally followed by continuous positive airway pressure (CPAP).

PSV is a form of partial ventilatory support that delivers a pre-selected amount of positive pressure in response to a spontaneous inspiratory effort. An inspiratory airway pressure between 3 and 14 cm H_2O can reduce the inspiratory work load by overcoming the imposed work due to air-flow resistance from the tracheal tube, breathing circuit, and ventilator circuit.[3,4] The addition of positive end-expiratory pressure (PEEP) to PSV results in bi-level airway pressure. PEEP improves pulmonary mechanics impaired from the loss of glottic function caused by the artificial airway,[5] overcomes the loss of end-expiratory lung volume that occurs when patients are supine or semi-upright,[6] prevents dynamic airway closure in patients with airway diseases,[7] and improves gas exchange.[8]

CPAP is a spontaneous mode of ventilatory support that delivers a positive airway pressure throughout the breathing cycle. When the gas flow during inspiration is higher than the patient's inspiratory flow, CPAP delivering a continuous flow can overcome the air-flow resistance from the tracheal tube and the breathing circuit,[9] and can improve pulmonary mechanics[5,6,10] and offset the intrinsic PEEP (or auto PEEP).[7]

Following prolonged mechanical ventilation and gradual weaning with PSV, some physicians discontinue mechanical ventilation directly, without additional modes of support. However, it is possible that the level of inspiratory airway pressure during PSV is greater than necessary to simply overcome the imposed work,[3] thus relieving the

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patient from a portion of the physiologic work of breathing (WOB), and as a consequence the patient can fail weaning. For this reason, some physicians extend weaning with CPAP.[11,12] However, the inspiratory airway pressure during CPAP may also be inappropriate— for instance, CPAP may be inadequate to overcome the imposed work,[13] resulting in additional work load and, therefore, unnecessary respiratory muscle fatigue. Thus, the possible benefits of CPAP can be offset by its drawbacks and there may be no overall advantage in extending weaning with CPAP.

The WOB is important in assessing the performance of the respiratory muscle activity and is a reliable predictor of weaning success.[11,14–16] If the WOB is too low, the ventilatory support may be high, relieving the patient from working during breathing.[3,17] If the WOB is too high, the patient is working harder than he would be if extubated or decannulated.[13] Comparing the measured WOB to published results aids in the determination of when the patient can be extubated or decannulated.[11,14–16]

Objectives

The goal of this study was to determine whether extending weaning with CPAP confers an advantage over removing patients directly from PSV and PEEP. First, we determined whether the WOB during PSV with PEEP was the same as during CPAP and appropriate for successful removal from mechanical ventilation. Second, we studied whether this earlier removal from mechanical ventilation resulted in a higher failure rate than extended weaning with CPAP.

Methods

Patients and Protocol

We studied 42 patients being weaned from mechanical ventilation. In our intensive care unit, the sequence of weaning after prolonged mechanical ventilation (> 72 hours) is as follows: gradual lowering of PSV during the daytime, with overnight rest on assist control ventilation using a Siemens Servo 900C.* While on PSV, we routinely employ 5 cm H_2O of PEEP, and set trigger sensitivity at –2 cm H_2O. This weaning process may take several days, until the pre-set inspiratory pressure level on PSV decreases to 5 cm H_2O (PSV_{5+5}, 5 cm H_2O PSV plus 5 cm H_2O of PEEP). At this point, some physicians choose to prolong weaning with 5 cm H_2O of CPAP ($CPAP_5$) for at least 24 additional hours. CPAP is provided by an adjustable Downs Flow Generator, a disposable corrugated plastic tube, a nonreservoir-humidified circuit, and an external PEEP valve. Either from $CPAP_5$ or from PSV_{5+5}, extubation or decannulation is the next step in our weaning algorithm. Intermittent face-mask CPAP is used for up to 24 hours in those patients who are extubated or decannulated from PSV_{5+5}.

All our patients entered the study after they were gradually weaned and when they were ready to be supported with an inspiratory pressure level of 5 cm H_2O and an external PEEP of 5 cm H_2O. Patients were included in the study when (1) the process that led to the patient's respiratory failure had improved; (2) the patient was hemodynamically stable and there was no further need for vasoactive agents; (3) the patient had adequate gas exchange (as indicated by an arterial oxygen tension $\geq$ 60 mm of Hg and fraction of inspired oxygen $\leq$ 0.40); (4) the patient could tolerate pressure support levels between 5 and 10 cm H_2O; (5) the patient could inspire with a peak negative pressure $\leq$ –20 cm H_2O; (6) the patient was judged by his or her attending physician to be able to continue the weaning. The University of Mississippi Investigation Review Board exempted this study from review.

Two modes, PSV_{5+5} and $CPAP_5$, were compared in all the patients, after they were rested overnight on assist-control mode. The Siemens Servo 900C was used for testing PSV_{5+5}, and the Downs Flow Generator was used for testing $CPAP_5$. Each patient was stable for a period of 20–30 minutes during the new ventilatory mode before data were collected. Modes were tested by pseudorandom order (by computer-generated numbers). The patients were rested on assist-control ventilation for 30 minutes between modes. Inspired gas was maintained at 30–40% oxygen, and humidified by a pass-over heater in both modes. Peak negative pressures were measured prior to data collection, and arterial blood gases were obtained on each mode immediately following the data collection.

Each patient was weaned from PSV_{5+5} or $CPAP_5$ at the discretion of his or her physician, who was not aware of study results. At our institution, some physicians always wean directly from PSV_{5+5} while others extend weaning with $CPAP_5$. Even though the assignment to the 2 groups in our study was not random, the treatment of any given patient depended solely on the patient's physician, whose call schedule was determined months in advance, and was independent of the patient population present in the intensive care unit at the time of the study.

All patients were weaned from PSV_{5+5} for approximately 24 hours, at which point, 20 patients (Group A) were removed from full ventilatory support. Of these 20 patients, 15 were extubated, 2 underwent decannulation with a tracheal button, and 3 had permanent tracheostomies (due to obstructive sleep apnea). Following extubation or decannulation, intermittent face-mask $CPAP_5$ was employed. Twenty-two patients (Group B) had their wean-

* Suppliers of commercial products are identified in the Product Sources section at the end of the text.

Table 1. Difference in Work of Breathing between PSV_{5+5} and $CPAP_5$ in 42 Patients

Group	PSV_{5+5}	$CPAP_5$	p value
All Patients (n = 42)	0.67 ± 0.54 (0.08)	0.96 ± 0.53 (0.08)	< 0.0001
Weaning success (n = 36)	0.64 ± 0.53 (0.09)	0.95 ± 0.52 (0.09)	< 0.0001
Weaning failure (n = 6)	0.90 ± 0.61 (0.25)	1.03 ± 0.65 (0.26)	0.0022
Group A (n = 18)	0.76 ± 0.62 (0.15)	1.01 ± 0.57 (0.14)	0.0097
Group B (n = 18)	0.51 ± 0.41 (0.10)	0.89 ± 0.47 (0.11)	< 0.0001

PSV_{5+5} and $CPAP_5$ values are means ± standard deviation, reported in joules per liter, with the standard error of the estimate shown in parentheses. The p values were calculated using the paired t test. PSV_{5+5} = pressure support ventilation of 5 cm H_2O with an external positive end-expiratory pressure of 5 cm H_2O; $CPAP_5$ = continuous positive airway pressure of 5 cm H_2O.

ing extended for an additional 24 hours with $CPAP_5$, at which point 13 of the Group B patients were extubated, 4 underwent decannulation with a tracheal button, and 5 had permanent tracheostomies (3 patients had obstructive sleep apnea, 1 patient had a cerebrovascular accident, 1 patient had tracheal stenosis). All patients were followed for 48 hours after the discontinuation of PSV_{5+5} and $CPAP_5$. Weaning from mechanical ventilation was considered successful if a patient did not require full mechanical ventilatory support within these 48 hours.

WOB Data Collection and Analysis

Measurements were made at the beginning of PSV_{5+5} to determine the WOB at a fixed point before the final stages of weaning. A report by Petrini et al suggested that the WOB would change little between this point and extubation or decannulation; in that report the WOB during a low but constant level of PSV was not different between 1 and 24 hours prior to extubation in patients gradually weaned with PSV following prolonged mechanical ventilation.[18]

The WOB per unit volume in joules per liter (J/L) was measured using a CP-100 Pulmonary Monitor. The flow transducer used for continual measurements of airway flow and airway pressure was placed at the proximal end of the endotracheal or tracheostomy tube. Esophageal pressure was measured with an esophageal balloon incorporated into a nasogastric tube. Balloon placement was checked in each patient by performing the airway occlusion test.[19]

Each data collection period was 5 minutes. Data were sent from the CP-100 Pulmonary Monitor to an IBM-compatible computer via a serial port and stored in the computer using software provided by BiCore. This pulmonary monitor reliably provides a breath-by-breath, real-time display of the measured and calculated weaning parameters, including WOB.[20] (Its use is described in a report by Nilsestuen et al.[21]) The pulmonary monitor assumes a normal chest wall compliance of 0.2 L/cm H_2O in the calculation of WOB. The value of the chest wall compliance for individual patients may have been different from normal; however, the chest wall compliance would have been the same during the 2 modes because they were tested sequentially in each patient. Thus, differences in the total WOB would have been due to differences in the work done by the lung. Data were analyzed using a Gaussian-breath-elimination method that we developed in order to omit breaths with artifactual changes in esophageal pressure due to nonrespiratory maneuvers.[22] The WOB in joules per minute (J/min) was calculated breath-by-breath in our data analysis program by multiplying WOB per unit volume by the minute ventilation.

Statistics

Prior to the beginning of the study, the minimum required sample size was calculated to be approximately 20 patients in order to detect a difference of 30% between the 2 modes in each group, using the paired t test, and assuming $p < 0.05$, a statistical power of 80%, and a WOB variability of 40%, as we usually found in our patients.

Data are reported as means, plus or minus the standard deviation (SD), with the standard error of the estimate (SEE) in parentheses. The SEE was used to estimate the accuracy of the determination of the mean. The SD was used to describe the distribution of the values in the test population.[23]

Comparisons between each mode were made by the paired t test. Between-group comparisons were made by the unpaired t test. Frequency of observations in the 2 groups was compared using the chi-square test (χ^2). Results were considered significant with a $p < 0.05$ for all tests.

Results

The mean WOB was less during PSV_{5+5} than during $CPAP_5$ when all 42 patients were taken together ($p < 0.0001$), as well as when the 36 patients who succeeded and the 6 patients who failed were considered as separate groups (Table 1). The WOB measured in J/min was also

Table 2. Respiratory Parameters in PSV_{5+5} and $CPAP_5$ Mode

Parameter	Weaning Success (n = 36)			Weaning Failure (n = 6)		
	PSV_{5+5}	$CPAP_5$	p value	PSV_{5+5}	$CPAP_5$	p value
WOB (joules/min)	7.66 ± 7.25 (1.21)	9.25 ± 7.29 (1.12)	0.05	9.17 ± 9.12 (3.73)	10.35 ± 9.39 (3.83)	0.13
V_T (L)	0.40 ± 0.13 (0.02)	0.35 ± 0.12 (0.02)	< 0.0001	0.43 ± 0.16 (0.06)	0.34 ± 0.13 (0.05)	0.001
f (breaths/min)	23.0 ± 6.4 (1.1)	24.2 ± 6.2 (1.0)	0.10	28.6 ± 4.4 (1.8)	29.4 ± 4.4 (1.8)	0.38
Auto-PEEP (cm H_2O)	3.7 ± 1.5 (0.3)	4.8 ± 1.2 (0.2)	0.002	3.4 ± 1.8 (0.7)	4.8 ± 0.3 (0.1)	0.15
T_I/T_{TOT}	34.6 ± 4.7 (0.8)	40.2 ± 5.9 (1.0)	< 0.0001	34.2 ± 2.5 (1.0)	39.6 ± 3.5 (1.5)	0.002

PSV_{5+5} and $CPAP_5$ values are means ± standard deviation. The p values were calculated using the paired t test. PSV_{5+5} = pressure support of 5 cm H_2O with an external positive end-expiratory pressure of 5 cm H_2O; $CPAP_5$ = continuous positive airway pressure of 5 cm H_2O; WOB = work of breathing; V_T = tidal volume; f = respiratory rate; Auto-PEEP = auto-positive end-expiratory pressure; T_I/T_{TOT} = duty cycle.

Table 3. Characteristics of the 42 Patients Studied

Characteristic	Group A*	Group B*	p value
Patients (n)	20	22	
Age (years)	46 ± 19	45 ± 16	0.85†
Gender (Male:Female)	8:12	12:10	0.53‡
ID of Tracheal Tube (mm)	8 ± 0.5	8 ± 0.5	0.2†
Tracheal Tube Type (n, ET:TT)	15:5	13:9	0.44‡
Duration of MV (days)	13 ± 13	13 ± 10	0.86†
Peak negative pressure (cm H_2O)	43 ± 18	40 ± 14	0.55†
P_{aO_2}/F_{IO_2} (mm Hg)	357 ± 83	339 ± 109	0.55†
Etiology of Respiratory Failure (n):			
Pneumonia	10	10	
Upper airway obstruction	3	2	
Chronic obstructive pulmonary disease	2	3	
Central nervous system insult	2	3	
Sepsis	1	2	
Cardiac arrest	1	0	
Cardiomyopathy	0	2	
Diabetic ketoacidosis	1	0	

*Mean ± standard deviation, †Calculated using unpaired t-test, ‡Calculated using the chi-square test, ID = internal diameter; ET = endotracheal tube; TT = tracheotomy tube; Duration of MV = duration of mechanical ventilation prior to entering the study; P_{aO_2} = arterial oxygen tension (in mm Hg); F_{IO_2} = fraction of inspired oxygen.

less during PSV_{5+5} than during $CPAP_5$ in patients who were weaned successfully (Table 2). There was no statistically significant difference in the WOB measured in J/min between PSV_{5+5} and $CPAP_5$ in the patients who failed weaning, though this result may be an artifact of the low number of failures, which limited the statistical power of this test comparison. Other respiratory parameters in weaning success and failure in each mode are shown in Table 2. In both weaning success and weaning failure, the tidal volume was lower and the duty cycle was longer with $CPAP_5$ than with PSV_{5+5}. There were no statistical differences in respiratory rates between PSV_{5+5} and $CPAP_5$ in either weaning success or weaning failure. Respiratory rates in all groups were < 30 breaths per minute. In patients who were weaned successfully, the presence of auto-PEEP was statistically different, but not clinically different, between PSV_{5+5} and $CPAP_5$, but in the patients who failed weaning there was no significant difference in the presence of auto-PEEP.

The 42 patients were divided into 2 groups. Group A (n = 20) was weaned from PSV_{5+5}. Group B (n = 22) was weaned first on PSV_{5+5} and then further extended with $CPAP_5$. There was no statistical difference between the 2 groups with regard to age, gender, size of artificial airway, duration of mechanical ventilation, peak negative pressure, or oxygenation (Table 3). The patients in both groups shared similar etiologies requiring the initial mechanical ventilation. In the group as a whole, WOB measurements in each group were less during PSV_{5+5} than during $CPAP_5$ (p = 0.0097 for Group A; p < 0.0001 for Group B. See Table 1). However, there were no statistically significant differences in the WOB measured during

PSV_{5+5} in the 2 groups: Group A = 0.76 ± 0.62 (0.15) J/L versus Group B = 0.51 ± 0.41 (0.10) J/L, p = 0.17, unpaired *t* test. Similarly, there were no statistically significant differences in the WOB measured during $CPAP_5$ in the 2 groups: Group A = 1.01 ± 0.57 (0.14) J/L versus Group B = 0.89 ± 0.47 (0.11) J/L, p = 0.52, unpaired *t* test.

A total of 18 patients in each group were weaned successfully. A total of 6 patients (2 patients in Group A and 4 patients in Group B) failed weaning (ie, required mechanical ventilation within 48 hours). There were no statistical differences between the failure rates in the 2 modes (2 of 20 in Group A versus 4 of 22 in Group B, χ^2, p = 0.75).

Discussion

In a group of 42 mechanically ventilated patients judged ready to be weaned, the WOB was higher with $CPAP_5$ than with PSV_{5+5}. This finding may be due to the different levels of airway pressure delivered by these 2 modes. PSV is a form of partial ventilatory support that delivers a selected amount of positive pressure in response to a spontaneous inspiratory effort, while CPAP is a spontaneous ventilatory mode where positive pressure is maintained throughout the breathing cycle. With PSV_{5+5}, the total inspiratory airway pressure is maintained at 10 cm H_2O, whereas during $CPAP_5$ the inspiratory pressure is 5 cm H_2O. In both modes the expiratory airway pressure is 5 cm H_2O. Thus, the higher WOB during $CPAP_5$ may reflect the greater inspiratory airway pressure provided by PSV_{5+5} versus $CPAP_5$. In addition, the greater inspiratory pressure during PSV_{5+5} can result in the increased tidal volume seen with PSV_{5+5} versus $CPAP_5$ (see Table 2).

Two other factors have been reported to contribute to the WOB. First, while the presence of auto-PEEP can increase the WOB,[24] in this study auto-PEEP was approximately 1 cm H_2O less in PSV_{5+5} than in $CPAP_5$ (see Table 2), and that difference is too small to explain the observed difference in WOB. Second, the ventilatory circuits for providing PSV[25] and CPAP[9,26] can impose different amounts of inspiratory work. However, we have previously shown that the circuitry used in this study results in similar WOB when providing similar levels of pressure.[27]

The pulmonary monitor assumes a normal chest wall compliance (C_{cw}) of 0.2 L/cm H_2O in the calculation of WOB. Patients with respiratory failure could have a C_{cw} as low as 0.1 L/cm H_2O. Although it is not likely that our patients had such a low chest wall compliance (since they had recovered from the respiratory failure and were being weaned), we calculate that, in our patients, a halving of C_{cw} (from 0.2 to 0.1 L/cm H_2O, which would double the chest wall work), would only increase the total WOB by no more than 20%, a value well within the variability of the measurement.

Recently $CPAP_5$ has been shown to increase the imposed WOB. Kirton et al[13] studied the WOB in 28 patients after the patients were placed on $CPAP_5$ for 20 minutes from minimal mechanical ventilatory support (ie, intermittent mandatory ventilation of 0–2 breaths/minute, PSV < 10 cm H_2O, PEEP of 5 cm H_2O). In 6 patients the WOB was < 0.8 J/L, while in the remaining 22 patients it was > 0.8 J/L. They measured imposed work and physiologic work in these 22 patients, and in all but one they found that the physiologic work was < 0.8 J/L. These 21 patients were subsequently extubated, while the remaining patient was not extubated, because of the high level of physiologic work. The respiratory rates were > 30 breaths/minute in all 28 patients, and this tachypnea was attributed to the imposed work in the 21 patients. The respiratory rates observed in the present study were < 30 breaths/minute, despite WOB values > 0.8 J/L with $CPAP_5$ (see Tables 1 and 2), and we did not observe any signs of distress. Since the failure rate in the 2 groups was not different, the respiratory muscle activity was most likely adequate to overcome any additional increase in work that the $CPAP_5$ might have imposed.

Two previous studies failed to establish a significant difference in WOB between similar bilevel airway pressures provided by PSV versus CPAP. Petros et al[11] evaluated WOB using the Hamilton Ventilator in 8 patients who had respiratory failure but who did not have chronic obstructive pulmonary disease. They found that the WOB (in joules per liter) was not different between PSV_{5+5} versus $CPAP_5$. Similarly, Sassoon et al[28] reported that in 9 patients with chronic obstructive pulmonary disease recovering from respiratory failure, the WOB (measured in joules per liter) during PSV_5 (using a Puritan Bennett 7200a) with 8 cm H_2O CPAP (demand valve system with Puritan Bennett 7200a) was not different from that measured during 8 cm H_2O CPAP (flow-by with Puritan Bennett 7200a or continuous flow system with Emerson water column CPAP system). The absence of significant difference observed in the studies by Petros and Sassoon may be the result of the different types of mechanical ventilators and CPAP systems used, the different patient groups studied, or a lack of statistical power due to small sample size.

There are several strategies for weaning from mechanical ventilation.[6] We try to maintain a balance between the beneficial effects of a period of total rest for the respiratory muscles recovering from fatigue that may have occurred during respiratory failure[29] and the harmful effects of prolonged inactivity that result in muscle weakness and atrophy.[17] In our intensive care unit patients are rested on full mechanical support until they have started to recover

from the underlying illness. Weaning with gradual PSV begins when the hemodynamics are stable, there is adequate gas exchange, and the patient is able to trigger the ventilator. Because the primary process leading to mechanical ventilation has not completely resolved prior to the institution of weaning, we cannot substitute $CPAP_5$ or a T-piece in one step. Thus, our results cannot be compared directly with those studies that employ a switch from full ventilatory support to $CPAP_5$ or T-piece in order to separate patients who can tolerate immediate weaning from mechanical ventilation[30,31] from those who will require gradual weaning from prolonged mechanical ventilation.

There were no differences in WOB during PSV_{5+5} and during $CPAP_5$ between the patients of Group A, in whom full mechanical ventilatory support was removed from PSV_{5+5}, and the patients of Group B, in whom weaning was extended for an additional 24 hours (see Table 1). The 2 groups were otherwise similar (see Table 3). Furthermore, extending weaning with $CPAP_5$ did not improve the outcome. Thus, the present study supports the idea that, following gradual weaning with PSV, further use of $CPAP_5$ offers no additional benefit, and that patients can be extubated or decannulated directly from PSV_{5+5}. This shorter protocol saves a day of weaning by eliminating the use of CPAP.

The present study has 2 limitations. The first is that the assignment of patients to the 2 groups was not truly random. The decision as to whether a patient's mechanical ventilatory support should be removed directly from PSV_{5+5} or extended with $CPAP_5$ was made by the physician caring for that patient; some physicians always extubate from PSV_{5+5}, while others deem it safer to extend the weaning with $CPAP_5$. However, the assignment to a particular group was the result of call schedules that had been determined months in advance and were independent of the patients in the intensive care unit at the time of the study. Thus, the bias that may have entered into the study from the group assignment should be minimal. Table 3 shows that the 2 groups were very similar in all parameters measured, so the 2 groups were well matched.

A second limitation is that weaning success in Group A may have been assisted by the fact that we used intermittent face-mask CPAP following extubation or decannulation from PSV_{5+5} in the 17 patients. Face-mask CPAP has been shown to reduce the incidence of reintubation following extubation from mechanical ventilation.[32] However, the purpose of this study was to assess whether extending weaning with CPAP offered any advantage over direct removal from ventilator support at PSV_{5+5}, and the need for face-mask CPAP postextubation or postdecannulation does not reduce the benefit of an earlier extubation or decannulation.

Conclusion

Our results show that the WOB during PSV_{5+5} was lower than during $CPAP_5$, and the weaning outcome was the same in patients weaned from PSV_{5+5} as in patients whose weaning was extended with $CPAP_5$. Extending weaning with $CPAP_5$ offered no benefit in these patients, but instead imposed an additional load on the ventilatory-musculature. Thus, during gradual weaning with PSV following prolonged mechanical ventilation, full mechanical ventilatory support can be removed directly from PSV_{5+5}.

PRODUCT SOURCES

Ventilator:
Servo 900C, Siemens-Elma AB, Sweden

CPAP System:
Adjustable Downs Flow Generator, #9250, Vital Signs Inc, Totowa NJ

External PEEP Valve:
#9005, Vital Signs Inc, Totowa NJ

Pulmonary Mechanics Monitor:
Model CP-100, BiCore Monitoring Systems, Irvine CA

Esophageal Balloon Catheter:
Smart Cath, BiCore Monitoring Systems, Irvine CA

Pneumotachometer:
VarFlex, BiCore Monitoring System, Irvine CA

Humidifier:
RCI Conchatherm III, Respiratory Care Inc, Arlington Heights IL

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Continuous and Expiratory Tracheal Gas Insufflation Produce Equal Levels of Total PEEP

Edgar Delgado RRT, Adelaida M Miro MD, Leslie A Hoffman RN PhD, Frederick J Tasota RN MSN, and Michael R Pinsky MD

BACKGROUND: Tracheal gas insufflation (TGI) used in conjunction with mechanical ventilation can increase total positive end-expiratory pressure (total PEEP). We tested the theory that TGI delivered throughout the entire respiratory cycle (c-TGI) increases total PEEP more than expiratory phase TGI (e-TGI). We also studied whether a pressure relief valve in the ventilator circuit could prevent increase in total PEEP during TGI. METHODS: Using an artificial lung model and pressure control ventilation, we studied the effect of c-TGI and e-TGI, with and without a pressure relief valve, and with and without maintenance of a constant minute ventilation ($\dot{V}_E$), at 3 different inspiratory-expiratory ratios. RESULTS: Under constant $\dot{V}_E$ conditions, the increase in total PEEP was equivalent with c-TGI and e-TGI. Without adjustments to maintain $\dot{V}_E$ constant, $\dot{V}_E$ increased during c-TGI and decreased during e-TGI. Under all conditions increasing the inspiratory-expiratory ratio increased total PEEP. CONCLUSION: When $\dot{V}_E$ is maintained constant, c-TGI and e-TGI produce equivalent levels of total PEEP. Failure to adjust the ventilator settings during TGI creates changes in ventilatory parameters that are unique to each delivery system. [Respir Care 1999;44(4):428–433] *Key words: tracheal gas insufflation, mechanical ventilation, barotrauma, ventilatory support.*

Introduction

Tracheal gas insufflation (TGI) was developed to aid in the reduction of alveolar distending pressures and yet maintain adequate gas exchange when treating patients with acute lung injury. When utilized in conjunction with mechanical ventilation, TGI can enhance carbon dioxide (CO_2) elimination for a constant minute ventilation ($\dot{V}_E$), potentially reducing ventilatory requirements.[1–3]

TGI involves an intratracheal catheter advanced to a position approximately 2 cm above the carina. A flow of gas then insufflates the airway with an oxygen mixture equivalent to that delivered by the mechanical ventilator. The major mechanism of CO_2 elimination is believed to be CO_2 washout from the anatomic dead space proximal to the catheter tip.[2–6] Another possible mechanism is enhanced distal gas mixing due to the turbulence created at high TGI catheter flow.[1,2,7–12] Since TGI improves the efficiency of CO_2 elimination, it is possible either to reduce mechanical ventilatory support by decreasing $\dot{V}_E$ and peak airway pressure (P_{peak}), or to keep $\dot{V}_E$ constant while increasing CO_2 elimination.[2–4,8,13]

Several methods of TGI delivery have been proposed. The simplest method is to deliver TGI continuously throughout the respiratory cycle (c-TGI). TGI can also be gated (using solenoid valves) to deliver gas during specified phases of the respiratory cycle. Regardless of the delivery method, adverse effects have been observed with TGI, including increases in P_{peak} and end-expiratory lung volume. The increase in end-expiratory lung volume reflects a dynamic form of hyperinflation, and is often referred to as auto-positive end-expiratory pressure (auto-PEEP).[14–16] It has been postulated that increases in P_{peak} and end-expiratory lung volume are greater during c-TGI because gas is delivered throughout the respiratory

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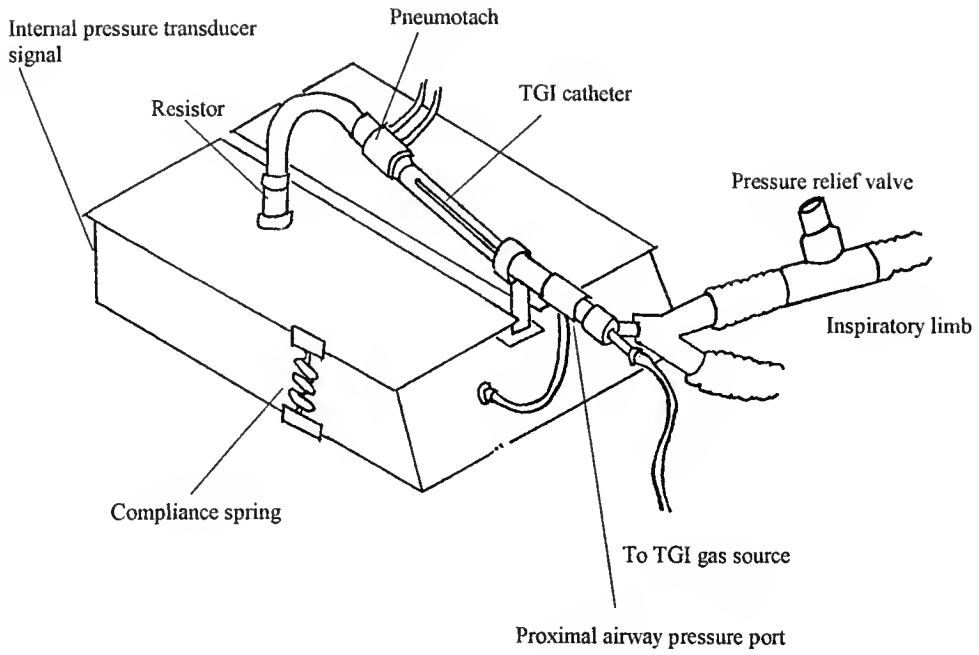

Fig. 1. Experimental apparatus. TGI = tracheal gas insufflation; pneumotach = pneumotachometer.

cycle.[8,15,16] Thus, a potential reason for the observed increase in auto-PEEP and consequent increase in total PEEP (set PEEP + auto-PEEP) with c-TGI is an increase in $\dot{V}_E$, because the ventilator-delivered tidal volume (V_T) was not proportionally reduced (volume control ventilation) or the ventilator circuit was not properly adapted to vent excess TGI volume (pressure control ventilation).[14,15]

There are several reports that describe a variety of interactions between TGI and mechanical ventilation that influence ventilatory parameters such as $\dot{V}_E$ and total PEEP.[14,15,17] When c-TGI is used with PCV, the ventilator initially compensates for the increased flow from the catheter by decreasing ventilator-derived V_T. However, this compensation ends if ventilator flow ceases. In this situation with c-TGI, gas continues to flow into the lung during the interval between cessation of ventilator flow and the beginning of expiration, which results in an increased volume of insufflated gas and, therefore, an increase in peak inspiratory pressure because the ventilator does not recognize the excess volume. This situation can be avoided by the insertion of a pressure relief valve into the ventilator circuit, which allows the excess gas to be vented into the atmosphere.[14] $\dot{V}_E$ is thereby maintained constant and an increase in pressure is avoided. Use of expiratory phase TGI (e-TGI) avoids this problem because gas flows only during expiration. However, Imanaka et al[18] recently found that, during PCV, e-TGI decreases $\dot{V}_E$ (compared to no TGI).

Both c-TGI and e-TGI produce back pressure during PCV and, thus, similarly increase expiratory resistance. But the effect of either TGI method on $\dot{V}_E$ and total PEEP is unclear. We hypothesized that as long as $\dot{V}_E$ was kept constant during PCV, total PEEP levels would be the same using either c-TGI or e-TGI. To test this hypothesis, we used a single compartment artificial lung model to measure end-expiratory lung pressures. We then compared total PEEP levels during c-TGI with those produced during e-TGI, with and without adjustments to maintain $\dot{V}_E$ constant.

Methods

Equipment, Calibration, and Initial Conditions

The apparatus consisted of a single-compartment artificial lung, linear resister, TGI catheter device, pneumotachometer for $\dot{V}_E$ measurement, adjustable pressure relief valve, and mechanical ventilator (Fig. 1).* The artificial lung model was operated in the single-lung mode with the TGI catheter (1.67 mm inner diameter) inserted into the artificial trachea (73 cm total length including pneumotachometer, resister, and TGI catheter swivel). A calibrated pneumotachometer, connected to a differential pressure transducer, was placed within the artificial airway 14.5 cm distal to the tip of the TGI catheter to record total $\dot{V}_E$ (ventilator + TGI) entering the lung. Volume calibration measurements were performed by injection of a known volume (0.750 L) using a calibration syringe. Volume calibration measurements were repeated until a $\leq$ 3% varia-

* Suppliers of commercial products are identified in the Product Sources section at the end of the text.

Table 1. Continuous Tracheal Gas Insufflation versus Expiratory Tracheal Gas Insufflation with and without Adjustments to Maintain Constant Minute Ventilation

	Control			Continuous TGI (pressure relief valve)			Expiratory TGI (increased driving pressure*)			Continuous TGI (no relief valve)			Expiratory TGI (no pressure adjustment)		
I:E ratio	1:2	1:1	2:1	1:2	1:1	2:1	1:2	1:1	2:1	1:2	1:1	2:1	1:2	1:1	2:1
$\dot{V}_E$ (L/min)	10.3	11.1	10.1	10.2	11.1	10.1	10.3	11.1	10.1	10.2	11.5	11.2	9.6	10.2	9.1
Total-PEEP (cm H_2O)	5.0	6.0	9.6	7.5	8.5	12.2	7.5	8.5	12.6	7.5	8.7	13.7	7.5	8.3	11.9
P_{peak} (cm H_2O)	36.0	39.4	41.0	38.5	42.1	43.8	38.5	42.2	44.6	38.5	43.7	49.2	36.2	39.6	40.6
P_{peak}–Total-PEEP (cm H_2O)	30.1	33.4	31.4	31.0	33.6	31.6	31.0	33.7	31.6	31.0	35.0	35.5	28.7	31.3	28.7
Paw (cmH_2O)	40.1	40.6	41.2	41.0	42.4	44.4	43.0	43.1	44.7	41.2	44.9	50.6	39.9	40.5	40.8

*Ventilator driving pressure increased to 38 cm H_2O during inspiratory-expiratory ratios 1:2 and 1:1, and increased to 39 cm H_2O during inspiratory-expiratory ratio of 2:1. TGI = tracheal gas insufflation; I:E = inspiratory-expiratory ratio; $\dot{V}_E$ = minute ventilation; PEEP = positive end-expiratory pressure; P_{peak} = peak intrapulmonary lung pressure; P_{peak}–Total-PEEP = calculation for the pressure gradient in the artificial lung; P_{aw} = proximal airway pressure.

tion end point was achieved. Resistance of the lung was set at 20 cm H_2O/L/S by insertion of the linear resister, and compliance was set at 0.02 L/cm H_2O by adjustment of the device's compliance spring.

Total PEEP and P_{peak} measurements were obtained from the pressure transducer inside the artificial lung and calibrated using known pressures as verified by a calibration analyzer. Proximal airway pressure (P_{aw}) was measured immediately distal to the ventilator Y connector. The mechanical ventilator was set in the pressure control mode with an initial inspiratory driving pressure of 35 cm H_2O, frequency of 16 breaths per minute, and PEEP equal to 5 cm H_2O to produce baseline control values (with no pressure relief valve in line). We used 5 cm H_2O PEEP because the compliance of our artificial lung model showed some alinearity at the initial opening of the compliance spring, which resolved with the addition of 5 cm H_2O of PEEP.

In each mode, measurements were taken at inspiratory-expiratory (I:E) ratios of 1:2, 1:1, and 2:1. The fraction of inspired oxygen was set at 0.21. After verification with an independent flow calibrating device, tracheal gas was insufflated at 10 L/min (the flow most commonly used in clinical practice at our institution). Expiratory TGI was delivered using an external solenoid valve electronically synchronized with the exhalation valve of the mechanical ventilator. During c-TGI and constant $\dot{V}_E$ conditions, a pressure relief valve was inserted into the inspiratory limb of the ventilator circuit and adjusted to vent excess volume to match control $\dot{V}_E$, per the experimental protocol. For e-TGI and constant $\dot{V}_E$ conditions, the ventilator inspiratory pressure setting was adjusted as needed to match the control $\dot{V}_E$. All our measurements were under ambient temperature and pressure, dry conditions. With repeated trials, pressures were constant within ± 0.2 cm H_2O, and volumes were accurate to ± 1%.

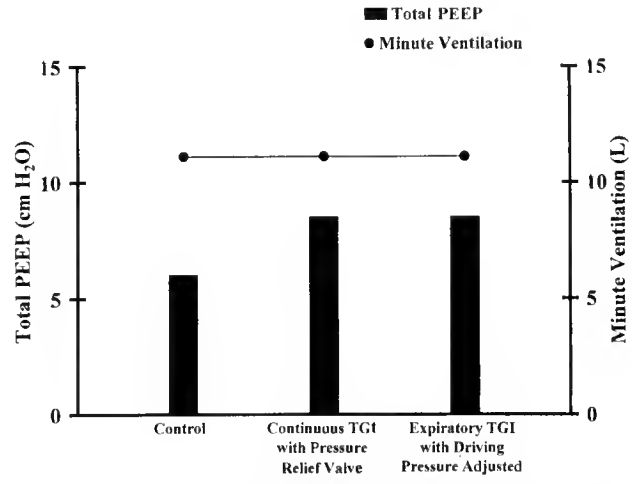

Fig. 2. Total PEEP and minute ventilation at an inspiratory-expiratory ratio of 1:1 with adjustments made to keep minute ventilation constant. PEEP = positive end-expiratory pressure; TGI = tracheal gas insufflation.

Protocol

Five experimental conditions were studied: (1) control (no TGI); (2) c-TGI with a pressure relief valve to maintain $\dot{V}_E$ constant; (3) e-TGI with an increase in ventilator driving pressure to maintain $\dot{V}_E$ constant; (4) c-TGI without a pressure relief valve; and (5) e-TGI without an increase in driving pressure. Each condition was tested using 3 I:E ratios (1:2, 1:1, 2:1).

Data Analysis

Because there was no measurable variance over multiple measurements, any differences between experimental conditions and control conditions were judged to be real, and no statistical analysis was performed.

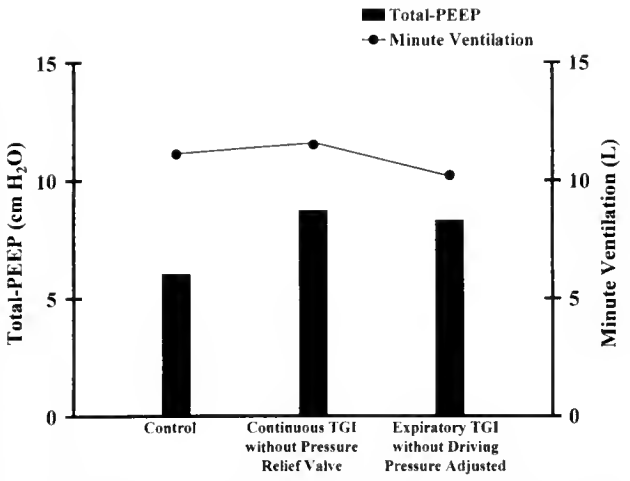

Fig. 3. Total PEEP and minute ventilation at an inspiratory-expiratory ratio of 1:1 without adjustments made to keep minute ventilation constant. PEEP = positive end-expiratory pressure; TGI = tracheal gas insufflation.

Results

Table 1 and Figures 2 and 3 summarize the results of our measurements.

Minute ventilation. $\dot{V}_E$ could be maintained constant between e-TGI and c-TGI, and when compared to control, but maintaining constant $\dot{V}_E$ required different maneuvers for c-TGI and e-TGI. $\dot{V}_E$ increased during c-TGI (10.2 L/min at an I:E ratio of 1:2; 11.5 L/min at an I:E ratio of 1:1; and 11.2 L/min at an I:E ratio of 2:1), but could be held constant by the insertion of a pressure relief valve into the ventilator circuit. Conversely, $\dot{V}_E$ decreased during e-TGI (9.6 L/min at an I:E ratio of 1:2; 10.2 L/min at an I:E ratio of 1:1; and 9.1 L/min at an I:E ratio of 2:1), but could be held constant by proportionally increasing the ventilator driving pressure.

Total PEEP. When $\dot{V}_E$ was held constant, the amount of TGI-induced total PEEP above control was equivalent for c-TGI and e-TGI:

7.5 cm H_2O versus 7.5 cm H_2O at an I:E ratio of 1:2;

8.5 cm H_2O versus 8.5 cm H_2O at an I:E ratio of 1:1; and

12.2 cm H_2O versus 12.6 cm H_2O at an I:E ratio of 2:1.

Pressure gradients between P_{peak} and total PEEP remained constant when $\dot{V}_E$ was held constant, reflecting a constant compliance in the system. As I:E ratio increased, total PEEP increased under all conditions (see Table 1).

Interactions between the ventilator system and TGI. If no modifications were made to the ventilator system, c-TGI increased $\dot{V}_E$ above control, while e-TGI decreased $\dot{V}_E$. These changes in $\dot{V}_E$ led to a greater total PEEP during c-TGI than during e-TGI, particularly at an I:E ratio of 2:1. Further, these changes in $\dot{V}_E$ corresponded to changes in the pressure gradient between P_{peak} and total PEEP (see Table 1).

Discussion

Tracheal gas insufflation can be an effective tool to promote CO_2 elimination from the anatomical dead space in both spontaneously breathing subjects[19] and during mechanical ventilation.[2,3,20] However, a problem limiting the successful application of TGI is the potential for over-pressurization of the airways and production of dynamic hyperinflation. Continuous TGI poses a greater risk of over-pressurization due to gas flow from the tracheal catheter during inspiration.[14,18] However, our study demonstrates that when $\dot{V}_E$ is maintained constant, both c-TGI and e-TGI produce equivalent levels of total PEEP. Furthermore, failure to make adjustments to the ventilator system during TGI creates changes in ventilatory parameters unique to each delivery system.

Continued interest in the clinical application of TGI has spurred debate regarding the optimal means to deliver this therapy. There are potential advantages and disadvantages to both c-TGI and e-TGI with regard to efficacy, safety, and ease of use,[21,22] but both techniques require an understanding of the unique interactions between TGI and the ventilator system, and of TGI's effects on ventilatory parameters. We designed our study to examine and describe the ventilator adjustments necessary to maintain constant $\dot{V}_E$ during PCV with TGI, and to document changes in ventilatory parameters when adjustments are not incorporated.

Prior research has evaluated auto-PEEP development with c-TGI during PCV and volume control ventilation (VCV),[15] and the use of a pressure relief valve during PCV with c-TGI to keep $\dot{V}_E$ constant.[14] Imanaka et al studied auto-PEEP development with e-TGI during PCV and VCV, as well as volume-adjusted c-TGI during VCV.[18] However, the difference in effect between c-TGI and e-TGI on total PEEP during PCV when minute volume is allowed to vary or is held constant has not been described.

Our data confirm that during PCV, both c-TGI and e-TGI increase total PEEP. As previously demonstrated by our group and others, the increase in total PEEP is due to a combination of an increase in airway pressure, limiting expiration, and delivery of a larger V_T.[1,14,15,18] Using c-TGI without volume adjustment increased $\dot{V}_E$, P_{peak}, and total PEEP when expiratory time was shortened (I:E ratios of 1:1and 2:1). However, when the inspiratory time was shortened, $\dot{V}_E$ and P_{peak} did not increase (as seen with an I:E ratio of 1:2). Using e-TGI without volume adjustment maintained P_{peak} but increased total PEEP. The resulting

decrease in the pressure gradient between P_{peak} and total PEEP decreased $\dot{V}_E$ at all I:E ratios, because, during PCV, the volume delivered depends on the pressure gradient between the proximal airway and the end-expiratory intrapulmonary pressure, which the ventilator does not measure. If the pressure gradient between proximal airway pressure and total PEEP decreases during e-TGI, delivered V_T decreases (as demonstrated by our data). Note, however, that the V_T decrease would be neither linear nor predictable from measures of inspiratory flow, peak inspiratory pressure, or ventilator inspiratory pressure setting. Further, the need to increase the inspiratory pressure setting to maintain the pressure gradient and V_T is not intuitively obvious from measurement of P_{peak} alone.

Increase in total PEEP during c-TGI with VCV is more easily addressed. In c-TGI with VCV, the increase in total delivered V_T (ventilator + TGI) results in increased $\dot{V}_E$. This c-TGI-induced increase in total V_T causes an increase in P_{peak} and total PEEP greater than that caused by e-TGI. To avoid the $\dot{V}_E$ increase associated with c-TGI, reduce ventilator-derived V_T by an amount equivalent to that delivered by TGI during inspiration.[2,21] After reducing ventilator-derived V_T, total PEEP levels with c-TGI during VCV are equivalent to those seen with e-TGI.[18] Our data demonstrate that if inspired $\dot{V}_E$ is kept constant by a pressure relief valve during c-TGI, or by increasing driving pressure during e-TGI, total PEEP levels are also equivalent during PCV.

Other factors can also increase auto-PEEP. Increased resistance due to the decrease in cross-sectional area of the endotracheal tube from the TGI catheter can increase auto-PEEP. Also, the expiratory time is inversely related to the amount of total PEEP formation, regardless of the mode of TGI delivery (as demonstrated by our comparisons at I:E ratios of 1:2 versus 1:1 versus 2:1). Clinically, this increase in total PEEP may result in increased alveolar dead space ventilation, which offsets TGI's benefits (the reduction in anatomic dead space and CO_2 washout). It is imperative to consider these phenomena when utilizing TGI with inverse ratio ventilation.

The lung model used in this study presents certain limitations. We used a single compartment model, which is remotely different from the complex animal or human model. However, this model permitted us to accurately measure true end-expiratory lung pressure (total PEEP), which may be impossible in the human lung. Since there are currently no techniques to clinically measure true alveolar pressures, titrating the ventilator system to proximal airway pressure may not match $\dot{V}_E$ conditions present at the alveolar level in the absence of TGI conditions. This limitation holds true for both c-TGI and e-TGI, and, therefore, the choice of method should be based on individual and institutional experience and preference.

Conclusion

Our study demonstrates that when $\dot{V}_E$ is maintained constant both c-TGI and e-TGI produce equivalent levels of total PEEP. It is necessary to adjust ventilator settings to maintain $\dot{V}_E$ constant during both c-TGI and e-TGI, and failure to adjust the ventilator system causes changes to ventilatory parameters unique to each delivery system. Despite recent advances in our knowledge of TGI, the challenges related to the monitoring of end-expiratory lung pressures, adequate humidification, and a pressure relief mechanism to address total endotracheal tube occlusion are yet to be resolved.

PRODUCT SOURCES

Artificial Lung Model:
Training Test Lung 2600I, Michigan Instruments Inc, Grand Rapids MI

TGI Catheter Device:
Portex Jet Ventilator Adapter #600101, Concord/Portex, Keene NH

Pneumotachometer:
No. 2 Fleisch, Lausanne, Switzerland

Linear Resistor:
112275 7100R20, Hans Rudolph, Kansas City MO

Adjustable Pressure Relief Valve:
#04230I, Bird Products Corp, Palm Springs CA

Mechanical Ventilator:
Puritan-Bennett 7200ae, Mallinckrodt, Pleasanton CA

Differential Pressure Transducer:
MP45, Validyne Engineering Corporation, Northridge CA

Independent Flow Calibrating Device:
Timeter Calibration Analyzer, Series RT 200, Allied Health Care Products Inc, St Louis MO

Calibration Syringe:
5540, Hans Rudolph, Kansas City MO

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Case Reports

Unrecognized Motor Neuron Disease: An Uncommon Cause of Ventilator Dependency in the Intensive Care Unit

Rodrigo Morales MD and Jorge E Mendizabal MD

A previously healthy woman developed ventilatory failure leading to ventilatory support. We ruled out primary cardiovascular and pulmonary etiologies for the ventilatory failure. Multiple attempts to wean ventilatory support were unsuccessful. Eventually, findings consistent with upper and lower motor neuron involvement were discovered, as well as paradoxical movement of the abdominal wall during inspiration. Nerve conduction studies and needle electromyographic examination showed signs of motor neuron degeneration such as those seen in amyotrophic lateral sclerosis (ALS). ALS should be considered a possible diagnosis in patients with otherwise unexplained ventilator dependence and paradoxical movements of the abdominal wall. [Respir Care 1999;44(4):434–436]
Key words: ventilatory dependency, motor neuron disease, amyotrophic lateral sclerosis, mechanical ventilation.

Introduction

Neurological disease is occasionally responsible for acute ventilatory failure requiring prolonged assisted ventilation in the intensive care unit (ICU). Motor neuron disease (MND), specifically amyotrophic lateral sclerosis (ALS) rarely presents with acute ventilatory failure due to weakness of the respiratory musculature. The diagnosis of ALS in these cases is usually made retrospectively and by eliminating other more common neurogenic or myogenic causes of ventilatory weakness. We present a case of ventilator dependence secondary to MND, and review the pertinent literature.

Case Report

A 70 year-old woman was transferred to our institution for ventilatory support after suffering a respiratory arrest. The patient had been complaining of "frequent falls" and progressive dysphonia for 1 week, but had noticed worsening of these symptoms in the 2 days prior to presentation. She denied dysphagia, sensory symptoms, muscle twitching, or cramps. On the day of presentation she complained of a headache and noted "twisting" of her mouth. Within minutes, while in the transferring facility, she became diaphoretic and progressed from mild dyspnea to frank ventilatory failure. After orotracheal intubation she was transported by emergency medical personnel to our hospital. Her medical history was unremarkable and she was a nonsmoker.

Physical examination revealed an alert and well nourished woman who was able to follow instructions while receiving assisted ventilation. She was afebrile, her vital signs were stable, and her initial cardiac and respiratory examinations provided no clues as to the cause of her respiratory arrest. Her neurological examination revealed mild generalized limb muscle weakness (strength 4/5), brisk deep tendon reflexes with unsustained bilateral ankle clonus and bilateral extensor plantar response. No muscular atrophy or limb or tongue fasciculations were noted, sensory examination was unremarkable, and anal sphincter tone was normal. A noncontrast computed tomography of the head (performed at the transferring facility) showed no acute findings, but revealed an incidental small right parietal convexity mass with the radiologic appearance of a dural based meningioma. A chest radiograph was unrevealing. Maximum inspiratory pressure (MIP), measured 24 hours after admission, was −8 cm H_2O [−0.784 kPa].

On hospitalization day 4 we set the ventilator to pressure support mode and recorded an MIP of −10 cm H2O [−0.980 kPa]. Over the following 24 hours we gradually decreased the pressure support; then the patient was extubated and placed on face mask. However, within 10 min-

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utes of extubation her oxygen saturation dropped and she became bradycardic. We began noninvasive ventilation with pressure support of 8 cm H_2O [0.784 kPa] and a positive end-expiratory pressure (PEEP) of 8 cm H_2O [0.784 kPa]. After several minutes the patient appeared exhausted and an arterial blood sample showed a pH of 6.95, P_{CO_2} of 113 mm Hg [15.1 kPa], P_{O_2} of 106 mm Hg [14.1 kPa], and a CO_2 content of 24 mmol/L. We reintubated the patient and again placed her on mechanical ventilation. Her course in the ICU involved further unsuccessful attempts to discontinue mechanical ventilation, eventually leading to a tracheostomy. During the course of her hospitalization the patient developed a nosocomial urinary tract infection, which was effectively treated with intravenous antibiotics. We eventually noted paradoxical movements of the abdominal wall during spontaneous inspiration, and a real-time fluoroscopically-guided sniff test revealed reduced but symmetrical diaphragmatic excursion. At that time MIP was −10 cm H_2O [−0.981 kPa], with a spontaneous tidal volume of 300 mL.

A consulting neurology team noticed mild facial diplegia and generalized weakness with distal atrophy of the interossei, hyperreflexia, and bilateral extensor plantar response. Their exam did not note fasciculations in the limbs or lingual musculature. Intravenous administration of 10 mg of edrophonium chloride (Tensilon) increased MIP by 5 cm H_2O [0.490 kPa]; the neurological team considered the test inconclusive. An assay for antibodies against the acetylcholine receptor in the muscle returned negative. A trial of physostigmine produced no improvement of her weakness, and we discontinued the physostigmine because it appeared to induce abdominal cramping and diarrhea. Muscle fasciculations occurred at this time and were initially attributed to anticholinergic toxicity from the physostigmine. Nerve conduction studies revealed small amplitude of the compound muscle action potential of the median, ulnar, and peroneal nerves, with preservation of the conduction velocity. Her sensory nerve action potentials were normal, and repetitive nerve stimulation did not show a decremental response. Needle electromyography (EMG) detected diffuse limb muscle denervation and neurogenic motor unit recruitment pattern. The findings were consistent with a diffuse motor neuronopathy such as that seen in ALS. The combination of upper and lower motor neuron disease identified in the physical examination and the results of the electrophysiological studies led to the diagnosis of ALS. The patient remained on ventilatory support and was eventually discharged to a ventilator-skilled nursing home facility.

Discussion

A primary neurological etiology is occasionally identified as the sole cause of ventilatory failure and/or ventilator dependence in the ICU setting.[1] The most common neurological causes of respiratory muscle weakness in the ICU are acute demyelinating polyradiculoneuropathy (Landry-Guillain-Barré syndrome), myasthenia gravis, critical illness polyneuropathy, acute intermittent porphyria, Lambert-Eaton myasthenic syndrome, and neuromuscular blockade.[1]

ALS belongs to a group of degenerative diseases collectively known as MND,[2] which include a spectrum of idiopathic degenerative diseases primarily affecting the motor neurons. MND can primarily affect the upper motor neuron (primary lateral sclerosis), the lower motor neuron (progressive spinal muscular atrophy), or both (ALS).[2] About 25% of cases present initially with bulbar symptoms characterized by the gradual onset of dysphagia, dysarthria, and dysphonia (primary bulbar sclerosis).[3] Respiratory muscle weakness, when it occurs, tends to present later in the course of ALS or primary bulbar forms of MND.[2,4] However, the majority of patients with MND suffer an insidious onset, with vague complaints of clumsiness, muscle twitching, cramping, and mild weakness.[2]

Previous reports describe ventilatory failure as the initial presentation of MND.[5–9] In all the reported cases, previously healthy individuals develop ventilatory failure leading to mechanical ventilation. The patient becomes ventilator-dependent because of profound neurogenic weakness of the respiratory apparatus. Paradoxical movements of the abdominal wall (as noted in our patient) are a sign of diaphragmatic weakness, and are suggestive of MND.[4,6,8]

Retrospectively, the history of our patient suggested possible involvement of the lower motor neuron bulbar musculature. Similarly, Chen et al[6] observed signs of upper motor neuron involvement prior to recognition of ALS in a series of MND patients presenting with ventilatory failure. Other reports of ALS as a primary cause of ventilatory failure do not describe signs of upper motor neuron involvement upon initial presentation.[5,7–9] The presence of muscle weakness is variable in these series, but note that signs of mild respiratory impairment are often reported in the months preceding the onset of overt respiratory failure.[5–9]

Nerve conduction testing in MND usually reveals reduced amplitude of the compound motor action potential, with a normal latency and conduction velocity in the absence of focal conduction block.[2] EMG of involved muscle groups shows evidence of spontaneous motor unit activity consistent with neurogenic denervation and reduced muscle unit recruitment patterns.[2] The diagnosis is usually confirmed if similar findings are recorded from 3 or more muscle groups from different limbs or the tongue.[2] Direct needle EMG study of the diaphragm is feasible in the ICU setting.[10] The technique for electrophysiological testing of the phrenic nerve and diaphragm is presented in detail by

Bolton.[10] In ALS, EMG needle examination of the diaphragm reveals spontaneous motor unit activity, with a decreased number of relatively normal-sized motor unit potentials.[10]

The choice among long term ventilatory management alternatives for ALS patients depends on the initial presentation of the ventilatory failure.[11] In ALS patients presenting with acute onset of ventilatory failure, tracheostomy is the most likely long-term option.[11] Chronic ALS patients, who suffer more insidious onset of ventilatory failure, can be managed with negative pressure or noninvasive mechanical ventilation, thereby reducing the need for specialized resources.[11] The invariably fatal outcome of ALS carries profound legal and ethical implications for the patients and their caregivers. In a recent large-scale survey of ALS patients, 56% admitted to considering assisted suicide at some point in their disease.[12] Psychological support and counseling services may help to alleviate their feelings of hopelessness.[12]

Summary

When more common medical or neurological etiologies cannot be established as primary causes of ventilatory failure or ventilator dependence, we recommend considering the diagnosis of MND. The presence of bulbar symptoms, signs of upper and lower motor neuron degeneration, or the occurrence of paradoxical abdominal movements during inspiration should prompt consideration of MND, and neurophysiological studies should be obtained in these cases.

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A 60-Year-Old Woman with Dyspnea on Exertion

Ali Emad MD

Case Summary

A 60-year-old woman sought medical attention because of a gradual increase in dyspnea on exertion. She denied chills, fever, weight loss, abdominal symptoms, chest pain, cough, expectoration, or other cardiopulmonary symptoms. Her medical history was negative for hypertension, diabetes, or trauma. The patient had no prior hospitalizations or recent medical evaluations. Physical examination revealed an obese woman who was afebrile, with a heart rate of 109 beats/min, a respiratory rate of 20 breaths/min, and blood pressure 110/70 mm Hg. Examination of the chest revealed bowel sounds in the lower portion of the left hemithorax. Heart sounds were normal, without murmurs or gallops. The abdomen was normal, as was the neurologic examination.

Figure 1 shows the posteroanterior and lateral chest radiographs. Arterial blood gas analysis showed pH 7.33, arterial carbon dioxide tension (P_{aCO_2}) 33 mm Hg, arterial oxygen tension (P_{aO_2}) 61 mm Hg, and oxyhemoglobin saturation 91% while the patient breathed room air (in Shiraz, Iran, elevation 1,600 m [5,000 ft], at normal barometric pressure 690 mm Hg). Chest fluoroscopy showed significantly restricted motion of the left hemidiaphragm, but no paradoxical motion on sniffing or coughing. Fiberoptic bronchoscopy showed mild narrowing of the left main bronchus and moderate to severe narrowing of the left upper and lower lobe bronchi due to extrinsic compression, and no evidence of endobronchial lesions. Cytologic and microbiologic studies were negative. An esophagogram and upper gastrointestinal series showed the esophagus and fundus of the stomach to be in normal position. Figure 2 shows a section from a computerized tomogram (CT) of the middle part of the chest. Abdominal sonography was normal. Barium enema showed displacement of the transverse colon into the left chest beneath the left hemidiaphragm.

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How would you answer these questions?

What are the major findings in the radiographs and CT?

What differential diagnosis is suggested by these findings?

What treatment is indicated?

Answers

Radiographs and CT. The left hemidiaphragm is markedly elevated. The mediastinum and heart are pushed to the opposite site, suggesting the presence of extrinsic pressure on these structures. Gas shadows are also visible. In the lateral chest radiograph, the leaf of the elevated left hemidiaphragm is completely visible and has a continuous line (arrow). All gas shadows are located under the leaf of the left hemidiaphragm.

The CT reveals intra-abdominal contents, including hollow-viscous structures and part of the spleen within the left hemithorax. There are no mediastinal, pulmonary, or diaphragmatic masses. The heart and mediastinum have been partly displaced to the right. The left lower lobe is collapsed. The right lung is well aerated.

Differential Diagnosis. Differential diagnoses for elevation of the left hemidiaphragm include: phrenic nerve palsy, atelectasis, subpulmonic effusion, subpulmonic abscess, diaphragmatic hernia, eventration of the diaphragm, and distended abdominal viscera.[1,2]

Phrenic nerve palsy is suggested by the presence of: elevation of the diaphragm; diminished, absent, or paradoxical motion of the diaphragm on inspiration; mediastinal shift on inspiration; paradoxical motion of the diaphragm upon sniffing or coughing during fluoroscopy,[3,4] and prolonged phrenic nerve conduction time detected by electrophrenic stimulation.[2] In this case, the absence of paradoxical motion of the left hemidiaphragm upon sniffing or coughing during fluoroscopy makes the diagnosis of phrenic nerve palsy unlikely.

Collapse of the left lower lobe can cause elevation of the left hemidiaphragm. However, there was no evidence of chronic pneumonitis, tuberculosis, or endobronchial lesion as the cause of atelectasis in this patient. Negative examinations of sputum, bronchial washing, and bronchial

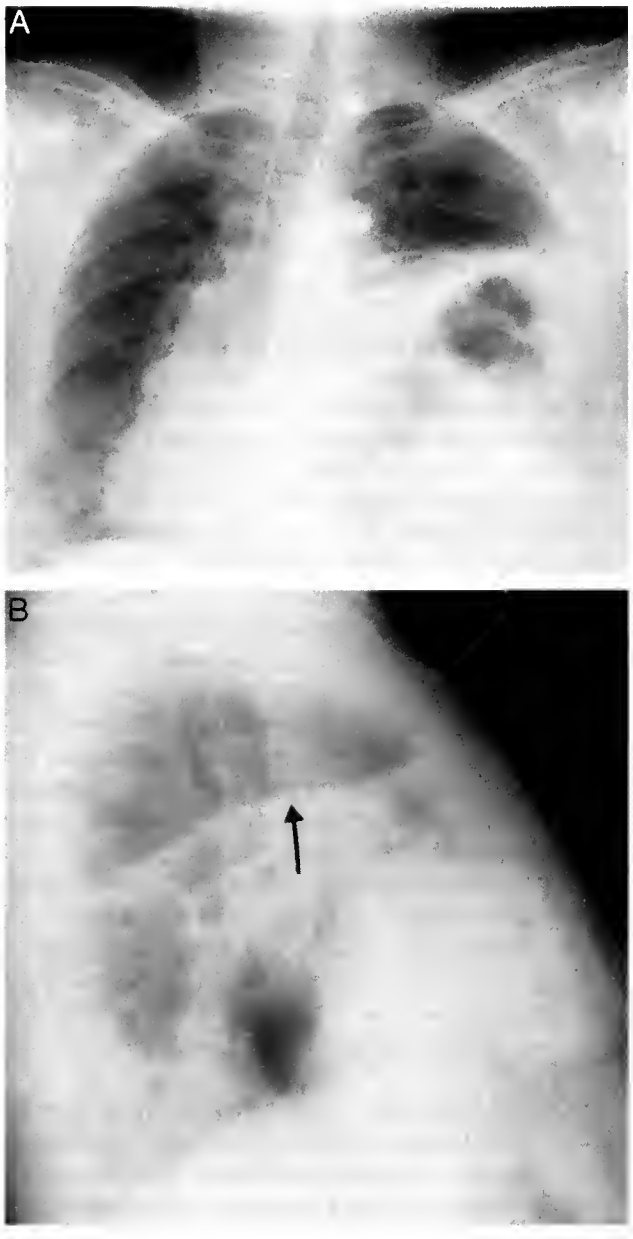

Fig. 1. Radiographs of a 60-year-old woman with respiratory distress. A. Posteroanterior chest radiograph. B. Lateral chest radiograph.

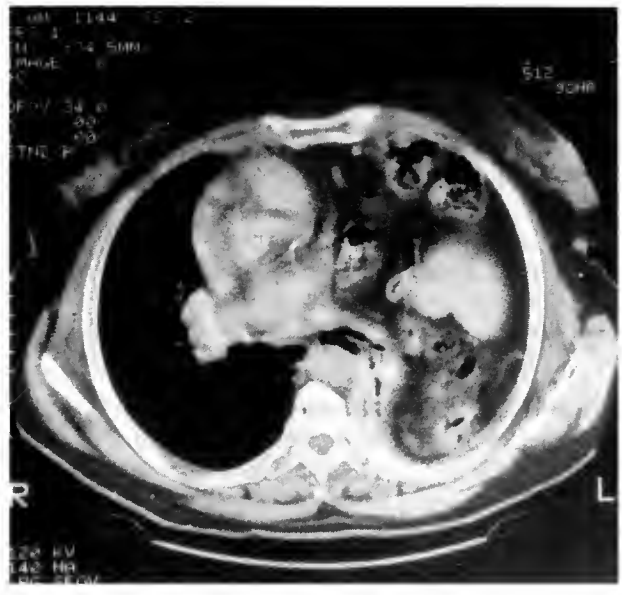

Fig. 2. Computed tomography (CT) image through the mid-portion of the patient's chest.

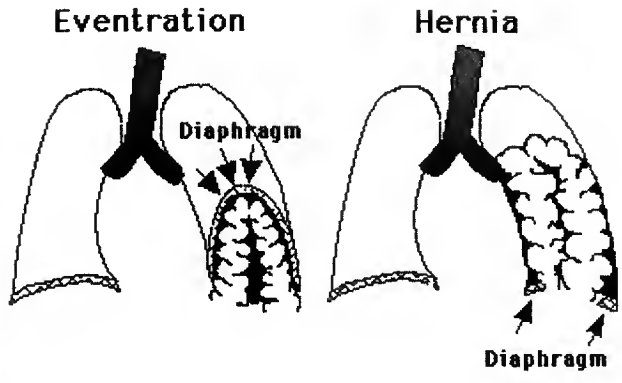

Fig. 3. Diagram showing the difference between diaphragmatic herniation and eventration. In diaphragmatic hernia the leaf of the diaphragm is seen as a broken line on the chest radiograph, while in eventration of the diaphragm it is seen as a smooth, unbroken line.

brushing suggest that the collapse of the left lower lobe is secondary to the elevation of the left hemidiaphragm rather than to intrinsic pulmonary disease.

In the absence of symptoms and signs referable to an abdominal process, and with a normal abdominal sonography, subpulmonic or subdiaphragmatic abscess or effusion can be excluded. The CT verifies the presence of extrinsic pressure on the lung tissue caused by the displaced abdominal contents into the left hemithorax.

The remaining 2 differential diagnoses are congenital diaphragmatic eventration and diaphragmatic hernia. Although the diagnosis of diaphragmatic eventration can be confirmed in most cases by routine chest radiograph and fluoroscopy, it can be difficult to differentiate from a diaphragmatic hernia, especially a hernia "sac." Figure 3 shows the difference between diaphragmatic eventration and diaphragmatic hernia with regard to the position and condition of the diaphragm.

A history of trauma to the lower chest or abdomen,[5] or the presence of a defect in the contour of the diaphragm on the chest radiograph[3,4] suggests diaphragmatic hernia, and this diagnosis might be confirmed by an upper gastrointestinal series or barium enema, since either the stomach or the colon can protrude through the diaphragmatic hernia.[5] Induction of a pneumoperitoneum is a safe and accurate

diagnostic procedure for differentiating a diaphragmatic hernia from a paralyzed or eventrated diaphragm. In diaphragmatic hernia, the injected air will enter from the peritoneum into the pleural cavity.[6] Sonography, radionuclide liver and spleen scanning,[7] and CT of the chest[8] may be necessary for diagnosis of hernia in occasional cases.

The diagnosis of severe eventration in this case was based on (1) the presence of an intact leaf of the left hemidiaphragm, best seen on the lateral chest radiograph as an unbroken line, (2) the lack of paradoxical motion of the diaphragm on fluoroscopy, and (3) the normal barium examination.

Treatment. In neonates and infants, complete eventration of the diaphragm is generally corrected surgically because the condition is potentially life-threatening.[9] Overall, symptomatic adult patients should primarily be managed with oxygen therapy and upright posture, though antibiotics may be necessary for infection control. If the patient responds well to supportive management, surgery might be avoided. Otherwise, surgery is required.[9] In patients with modest diaphragmatic elevation, and in those who demonstrate little functional impairment from the condition, surgery can be postponed. Surgery is not indicated for treatment of partial eventration, except when it is difficult to differentiate it from a mass lesion in the lung or when it causes pronounced symptoms.[10]

Surgical intervention for correction of complete congenital eventration is more commonly indicated in the pediatric age group than in adults. Asymptomatic patients should have regular follow up,[11] and may require surgical treatment if they develop dyspnea. If significant lung atelectasis is associated with eventration, surgery should be considered.[11,12] Because of her respiratory symptoms and the collapse of the lung, surgical treatment was offered to this patient, but was declined. Unfortunately she missed subsequent appointments and no follow-up examinations or other data are available.

Discussion

Eventration of the diaphragm, which is a rare condition, is broadly defined as an abnormally high position of part or all of the diaphragm. It occurs as a result of paralysis, aplasia, or atrophy to varying degree of the diaphragmatic muscle fibers.[10] There are 2 distinct etiologic types of eventration, congenital (nonparalytic) and acquired (paralytic).[10,13] Anatomically, the congenital eventrations are further divided into 3 forms: partial, complete, and bilateral.[12–15]

During weeks 8 to 10 of fetal life, the membranous diaphragm develops by fusion of the septum transversum and the dorsal mesentery of the foregut, and thereafter divides the coelomic cavity into the pleural and peritoneal cavities. The muscular element of the diaphragm is formed from the cervical myotomes.[2–4] Congenital eventration results from incomplete or absent muscularization of the pleuroperitoneal membrane.[7] Early return of the midgut to the abdominal cavity may be an important factor in the failure of normal diaphragm development.[1,7,10] The phrenic nerve is normal.

Eventration may be associated with other congenital abnormalities, such as aplasia of the lung, hypoplasia of the aorta, transposition of the abdominal contents, cleft lip, cleft palate, pulmonary sequestration, or bony abnormalities of the sternum, ribs or vertebrae.[7,10]

The most common location for the partial diaphragmatic eventration is the anteromedial portion of the right hemidiaphragm.[1,4] Characteristically, it is seen on the chest radiograph as a homogeneous, smoothly marginated soft tissue mass that is continuous with the diaphragm.[4] Barium esophagogram, gastrointestinal series, echocardiography, radionuclide liver scan, sonography, magnetic resonance imaging, and CT of the chest may be required to exclude other causes.[16] The majority of these patients are asymptomatic.[10]

Total or complete congenital eventration is usually found on the left side. Many patients with this condition remain asymptomatic.[10] Prominent symptoms are usually seen in infants because of shifting of the labile mediastinum.[10] Symptoms can arise from the abdomen, the respiratory system, or the heart. Abdominal symptoms, including nausea, vomiting, belching, and abdominal pain, are present in some patients. Volvulus of the stomach[17] and of the colon[18] can occur and are occasionally life-threatening. Cough, respiratory distress, cyanosis, and chest pain can occur due to compression of the lung or mediastinum.

It is important to note that the motion of the eventrated diaphragm may be normal, restricted, or absent. True paradoxical motion is not present in congenital (nonparalytic) eventration.[4]

Acquired (paralytic) diaphragmatic eventration follows injury to the phrenic nerve or its roots, which can be caused by trauma, childbirth (especially with breech presentation), or major abdominal surgery.[19] Central neurological disease, myopathies, and changes in diaphragmatic levels caused by neighboring lesions can also produce the acquired form of eventration. Acquired diaphragmatic eventration can be associated with aneurysm of the aorta, subdiaphragmatic hydatid cyst, pericarditis, alcoholic neuritis, spondylitis, and poliomyelitis.[19,20]

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Measurement of FEV_1 Using the Modified Spirometry Technique

James K Stoller MD, Daniel Laskowski RPFT, and Kevin McCarthy RCPT

Case Summary

A 72-year-old white male ex-smoker with a history of chronic obstructive lung disease presented with the complaint of increasing shortness of breath. A spirometry test was obtained using 2 different methods for coaching expiratory effort. Table 1 shows the Spirometry results. Figure 1 shows volume-time tracings for Spirograms 1 and 2. What would account for the differences in the results of these tests?

Table 1. Spirometry Results

	Predicted	LLN	Spirogram 1		Spirogram 2	
			Measured	Percent of Predicted	Measured	Percent of Predicted
FEV (L)	4.13	3.01	2.59	63	3.23	78
FEV_1 (L)	3.17	2.33	1.01	32	1.05	33
FEV_1/FVC	0.77	0.68	0.39	—	0.33	—
PEF (L/s)	7.66	5.30	5.58	73	6.24	82
$FEF_{50\%}$	3.59	1.96	0.35	10	0.29	8
FET (s)	—	—	9.39	—	17.6	—

Predicted—Mean predicted values as per Crapo et al.[1]
LLN = Lower limit of normal. The value as the lower 5th percentile of the normal range.
FVC = Forced vital capacity.
FEV_1 = Forced expiratory volume in 1 second.
FEV_1/FVC = Ratio of FEV_1 to FVC.
PEF = Peak expiratory flow (in liters/s).
$FEF_{50\%}$ = Forced expiratory flow at 50% of the FVC.
FET = Forced expiratory time (seconds).

Discussion

Based on the decreased FEV_1/FVC ratio (ratio of forced expiratory volume in 1 second to forced vital capacity), both spirograms indicate severe obstructive lung disease characterized as stage III chronic obstructive pulmonary disease (COPD) (ie, $FEV_1 < 35\%$ of predicted) by American Thoracic Society criteria.[2]

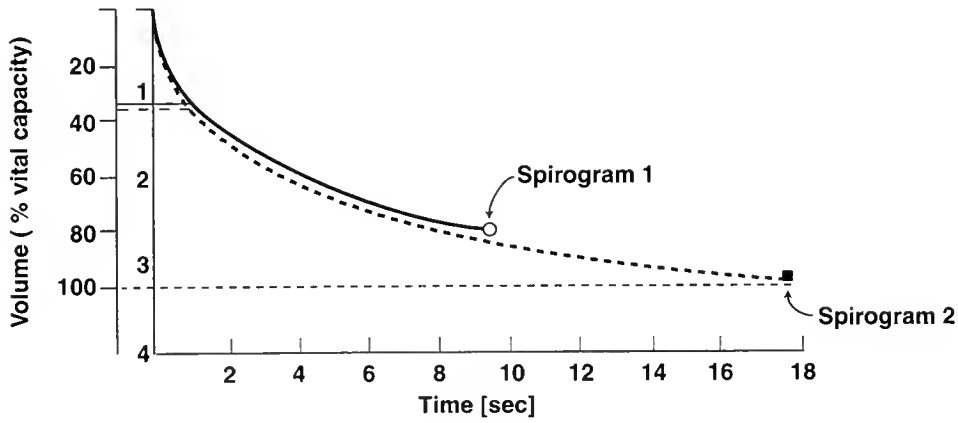

Fig. 1. Spirometry volume-time results.

The American Thoracic Society standardization of spirometry statement includes recommended criteria for both acceptability and reproducibility, which should both be satisfied.[3] Individual spirograms are acceptable if they:

1. are free from artifacts such as cough or glottic closure during the first second of exhalation, variable effort, obstructed mouth piece, or a leak,

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2. have good starts (time to peak flow of < 120 ms) or back extrapolated volume less than 5% of FVC or 0.15 L, whichever is greater,

3. and have a satisfactory exhalation time and meet end-of-test criteria (6 seconds of exhalation, a plateau in the volume-time curve, or if the subject cannot or should not continue to exhale).

The start-of-test criteria are best assessed on a flow-volume curve, whereas the end-of-test criteria are best assessed on a volume-time curve. After 3 acceptable spirograms have been obtained, the following reproducibility criteria should be applied: The 2 largest FVC and the 2 largest FEV_1 should be within 0.2 L of each other. If both the acceptability and reproducibility criteria are not met, continue testing with additional spirograms until either (1) the criteria are met, (2) a total of 8 tests have been performed, or (3) the patient cannot continue.

Spirogram 2 satisfies the end-of-test criteria, and the higher FVC is because of a more sustained expiratory effort.[4] The results of Spirogram 1 raise the issue of concomitant restrictive lung disease, because the FVC is below the lower limit of normal for our laboratory. The FVC in the second set of results is normal. Though the FEV_1 is similar in both spirograms, the higher value of the FEV_1/FVC ratio in the first effort results from the lower FVC value caused by early termination of the expiratory effort.

Satisfying end-of-test criteria for spirometry is difficult for patients with severe COPD because air flow limitation slows lung emptying. As a result, an expiratory plateau is infrequently achieved, and end-of-test criteria are satisfied only if a patient maintains an expiratory effort for 15 seconds. Although measuring the FEV_1 is paramount for clinical purposes and is unaffected by events after the first second of expiration, comparison of FEV_1/FVC ratios or FVC values in clinical and epidemiologic studies requires assuring adequate lung emptying or, in conditions of expiratory flow limitation, a standardized duration of expiratory effort.

To allow patients with severe obstructive lung disease to better achieve adequate expiratory duration and to satisfy end-of-test criteria, a modified expiratory maneuver for spirometry testing has been described by Stoller et al.[5] Specifically, the modified expiratory maneuver consists of a maximally forceful initial effort, followed by a relaxed expiratory effort begun on the technician's prompting at approximately 3 seconds (ie, when the expiratory flow falls below 200 mL/s). The impact of this modified expiratory maneuver on the rate of achieving end-of-test criteria by patients with COPD was examined in a crossover trial in which 48 subjects used a standard expiratory technique (ie, the technician encourages maximum forceful blowing for the entire duration of expiration) versus the modified expiratory technique. In this study, use of the modified technique was associated with a significantly higher rate of achieving end-of-test criteria (58% vs 19%, $p < 0.001$). This improved rate of satisfying end-of-test criteria was achieved without significant lessening of FEV_1 values. On the basis of these advantages, as well as improved comfort and patient preference, the modified expiratory technique has gained popularity and is the standard technique used in the Pulmonary Function Laboratory at The Cleveland Clinic Foundation.

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A Patient with Dyspnea and Acid Maltase Deficiency

Salim Kathawalla MD and Muzaffar Ahmad MD

Case Summary

A 39-year-old male with known history of acid maltase deficiency complained of chronic and progressive exertional dyspnea. The dyspnea was worse in the supine position and relieved by the upright posture. The results of physical examination and chest radiography were normal. Spirometry results are shown in Table 1. Based on the clinical findings and spirometric results, the patient underwent another diagnostic test.

1. What do the spirometric results show?
2. What was the additional diagnostic test that clarified the cause of this patient's dyspnea?

Discussion

The spirometry results indicate severe restrictive pulmonary impairment. Since the diffusing lung capacity is normal, a restrictive parenchymal lung process such as pulmonary fibrosis is unlikely. With the patient's history of dyspnea in the supine position and of acid maltase deficiency, known to cause respiratory muscle weakness, the spirometric findings suggest diaphragmatic weakness. The diagnostic test performed was a spirometry in the sitting and the supine position, which demonstrated a 50% decrease in vital capacity (VC) on assuming the supine position. A diagnosis of diaphragm paralysis was made and the patient was treated with a nocturnal rocking bed.

The characteristic abnormality of inspiratory muscle weakness is a low VC with a reduced total lung capacity and preserved residual volume. Carbon monoxide diffusing capacity is reduced, but is normal or raised when adjusted for volume.[2] This feature is useful to distinguish muscle weakness from alveolar disorders such as pulmonary fibrosis. VC by itself as a measure of respiratory muscle weakness is nonspecific and nondiagnostic. Also, in longstanding respiratory muscle weakness, the decrease in VC may be due to more than muscle weakness alone because of decreased chest wall and lung compliance, which is associated with microatelectasis.[3] Thus, VC by itself is not useful in monitoring patients with respiratory muscle weakness. The most widely applied tests for global inspiratory and expiratory muscle strength are static maximal inspiratory and expiratory pressure measured at the mouth (MIP and MEP). MIP values greater than 80 cm H_2O exclude clinically important respiratory muscle weakness.

In patients with neuromuscular disease and diaphragm weakness, there is a large decrease in VC upon assuming the supine position. However, even in normal young subjects there is a reduction in VC in the supine position, caused by a shift of blood to the pulmonary circulation, with a consequent reduction in intrathoracic gas volume, and a change in resting diaphragmatic position when su-

Table 1. Spirometry Results

Test	Sitting		Supine		Percent change in measured values
	Measured	Percent of predicted	Measured	Percent of predicted	
FVC (L)	1.34	27	0.67	14	−50
FEV_1 (L)	1.12	28	0.54	13	−52
FEV_1/FVC	0.84	102	0.81	99	−4
PEF (L/s)	4.79				
$FEF_{25-75\%}$	1.41	34			
FET (s)	8.92				
PIF (L/s)	2.16				
$FIF_{50\%}$ (L/s)	2.08				

Arterial blood gases on room air: pH 7.37, P_{aCO_2} 50 mm Hg, P_{aO_2} 80 mm Hg, bicarbonate 28 mEq/L, O_2 saturation 93%.
Predicted values = Mean predicted values per Crapo et al.[1]
FVC = Forced vital capacity.
FEV_1 = Forced expiratory volume in 1 second.
FEV_1/FVC = Ratio of FEV_1 to FVC.
PEF = Peak expiratory flow (in liters/s).
$FEF_{25-75\%}$ = Forced expiratory flow between 25% and 75% of the FVC.
FET = Forced expiratory time (seconds).
PIF = Peak inspiratory flow (in liters/s).
$FIF_{50\%}$ = Forced expiratory flow at 50% of the FVC.

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pine.[4,5] Further, in patients with restrictive and obstructive lung disease without diaphragmatic weakness, there is also a substantial decrease in VC upon assuming the supine position.[6] In a study of patients with neuromuscular disease and diaphragmatic involvement, all subjects had restrictive pulmonary impairment and VC decreased by 37–55% (mean of 46%) upon assuming the supine position.[7] In a study by Allen et al, a VC decrease of ≥ 25% upon assuming the supine position, in conjunction with a restrictive defect on lung function of over 35% with an obstructive defect, was very suggestive of diaphragmatic weakness.[6]

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Letters

Response to Lung Protective Ventilatory Strategies for ARDS

I read with great interest the point-of-view article by Kacmarek and Chiche concerning the use of lung protective ventilatory strategies (LPVS) for the acute respiratory distress syndrome (ARDS) published in the September 1998 issue of RESPIRATORY CARE.[1] I say "great interest" because I hold great enthusiasm for, and consider myself a proponent of LPVS in ARDS. However I am less enthusiastic and rather skeptical about the ability of LPVS to improve mortality in patients with ARDS. My skepticism is founded on the fact that the majority of ARDS nonsurvivors do not die from respiratory failure,[2] and despite my shared concerns with the authors that injurious ventilation may in fact potentiate translocation of proinflammatory cytokines and bacteria from the lung, I feel it is premature to attribute the absence of worsening multiple organ function to a ventilation strategy. In addition, work by Gattinoni et al[3] suggests that ARDS potentiated from a pulmonary insult may have more parenchymal consolidation that is less responsive to positive end-expiratory pressure (PEEP) than ARDS potentiated by extrapulmonary illness, which may be composed of more PEEP-responsive alveolar flooding and collapse, and perhaps increased nonpulmonary thoracic elastance. It may be ironic, therefore, that we may tailor ventilator settings to lung mechanics with the intention of improving survival, but those patients with better, more responsive mechanics (eg, ARDS triggered by sepsis) may more frequently die.[2] In terms of the Amato LPVS trial,[4] it is true that the LPVS study group had a better survival rate after 28 days, but there was no difference in survival between the study and control groups at hospital discharge. I remain optimistic that utilization of LPVS for ARDS may prove beneficial in the treatment of ARDS, however these benefits may more likely be manifested by a reduction in ventilator and intensive care unit days rather than a significant improvement in overall survival.

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Books, Films, Tapes, & Software

Physiological Basis of Ventilatory Support, John J Marini MD and Arthur S Slutsky MD, Editors. (Lung Biology in Health and Disease, Volume 118, Claude Lenfant, Executive Editor) Hardcover, illustrated, 1,464 pages. Marcel Dekker Inc, New York NY; 1998. $295.00.

A sentence from the preface establishes the rationale for this exceptional volume: "It is our view that broad-based and intimate knowledge of the physiology underpinning ventilatory support is indispensable to optimal care of difficult patients who require ventilatory assistance." In support of this belief, Drs Marini and Slutsky have compiled what I believe to be the best single source on the science of ventilatory support. The contributing authors include a nearly complete assembly of the most prominent clinician-scientists in this field. The range of topics covered is comprehensive, and the majority of the authors have addressed their assigned topics in a sophisticated and critical manner. As could be anticipated, there is some overlap among the chapters, but generally it is not truly redundant, because each chapter maintains its own assigned focus. The 38 chapters are arranged in 4 parts: the physiology underlying ventilatory support, the consequences of mechanical ventilation, implementing ventilatory support, and specific problems in ventilation.

This is not a book for beginners. The majority of the chapters are addressed to experienced clinicians with a well-established understanding of respiratory physiology. For teachers and investigators in the field of ventilator support of patients with respiratory failure, this book is a well-compiled summary of current opinion. By their choices of authors and topics, the editors have clearly succeeded in their goal to provide "a resource that will serve as a firm basis from which to advance our continually evolving knowledge."

Part One, on the physiology of ventilatory support, represents both the best and most challenging part of the book, exemplified by the first chapter, by Younes and Georgopoulos, on the neurophysiology of patient-ventilator interactions. This chapter makes a persuasive case for the benefits of proportional assist ventilation, although it does not cover specifics about the technique. Among the other excellent contributions in this part, the use of diagrams in the Loring chapter on mechanics of lungs and chest wall, and the Sassoon and Mahutte chapter on work of breathing are particularly helpful to readers reviewing those physics-based concepts. The work of breathing chapter also includes a useful discussion of the various approaches to the estimation of work of breathing by mechanically ventilated patients. The gas exchange chapter is disappointing only because the authors have previously written in-depth discussions of the topic at a higher level of sophistication, which would have been more appropriate for this book. The final section of the chapter by Russell is a scholarly critique of the practice of pharmacological maximization of oxygen delivery as a primary goal in the care of acute respiratory distress syndrome patients. It should be required reading for all those who remain believers in this intervention.

Part Two, on the consequences of mechanical ventilation, is more approachable for full-time clinicians than the first part. The lead-off chapter by MacIntyre provides a full discussion of patient-ventilator dyssynchrony with all of the standard ventilatory modes. It is followed by a scholarly review by Tremblay et al both of animal studies and of clinical investigations of ventilator-induced lung injury. That information is helpful background for interpreting the debate over which tidal volumes may be too high or which lung inflation volumes may be too low for acutely injured lungs. The chapter covering the anatomic consequences of endotracheal intubation is complete and exceptionally well done. It reminds even experienced clinicians of the substantial range of anatomic complications that can arise as a consequence of establishment of an artificial airway. The one disappointment of Part Two was the chapter on sedation, which includes a cookbook-style naming of agents that could be used, without a critical assessment of the goals and complications of sedation of a mechanically ventilated patient.

Part Three, on implementing ventilatory support, begins with an excellent chapter on the physiologic consequences of endotracheal intubation, which might more logically have been paired with the earlier chapter on the anatomic complications. The chapter on monitoring the mechanically ventilated patient has compiled a very useful review of all of the measurements that can be taken on a ventilated patient, including comments on the accuracy and interpretations of the measurements themselves. The chapter on full ventilatory support focuses primarily on applications of pressure control modes, which is appropriate for the general advanced level of the book. In addition, it provides a concise perspective on sedation and paralysis that is superior to the earlier chapter devoted to that topic. The companion chapter on partial ventilatory support again discusses the issues of patient-ventilator dyssynchrony, and includes a good overview of the merits of proportional assist ventilation to address this problem. Part Three includes chapters discussing other ventilatory support techniques, including applications of positive end-expiratory pressure and continuous positive airway pressure in severe air flow obstruction, the use of nitric oxide and other pharmacological approaches, noninvasive positive pressure ventilation, high-frequency ventilation, liquid ventilation, and extracorporeal support. These chapters recall the wide range of solutions that have been explored toward the goal of improving outcomes of patients with respiratory failure.

Part Four, on special problems in ventilation, includes an extensive review of barotrauma that features a complete discussion of the barotrauma mechanism first proposed by CC Macklin. This chapter discusses tidal volume-related lung injury unrelated to barotrauma, and addresses the more controversial issue as to whether ventilator-induced lung injury can produce systemic effects. Comparative evaluation of ventilator management strategies is one of the most difficult aspects of the care of patients in critical illness settings. While the obvious goal of these strategies is to optimize recovery, the variability of clinical conditions leading to ventilatory failure ordinarily force the choice of a surrogate end point that may not necessarily be linked to the quality of the eventual outcome.

The final chapter by Morris and Cook on clinical trials in mechanical ventilation is a reminder of the difficulties posed in obtaining interpretable data on strategies for ventilator management. It is a fitting final chapter for a book filled with things that we can do, asking the question, "How can we best investigate what we should do?"

While any suggestion for an addition to a book that is already over 1,400 pages long is not likely to be welcome, proportional assist ventilation does not receive the depth of coverage that it merits. Its physiologic rationale is unique, its application requires a strong grasp of individual patient physiology, and I believe it promises to be the most significant innovation in ventilator support of the past 2 decades. While it is discussed in 2 of the chapters, proportional assist ventilation deserves a chapter of its own in a volume devoted to the physiologic basis of ventilatory support.

It is a pleasure to encounter a well-edited and critically written volume intended for the most physiologically-oriented of respiratory practitioners. Again, this is not a book for beginners. Indeed, some of the initial chapters on the physiology of ventilatory support presume a higher level of physiologic sophistication than that possessed by most board-certified critical care physicians. Subsequent chapters on the clinical application of ventilatory support assume familiarity with all of the ventilatory support approaches used by modern intensive care unit practitioners, and hence will be difficult going for a physiologist with medical interests. Regardless, because of the high level of presentation covering a broad range of issues, this volume merits a prominent place on the bookshelves of every investigator and teacher working in the field of respiratory care.

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Mechanical Ventilation Manual, Suhail Raoof MD and Faroque A Khan MB, Editors. Soft-cover, illustrated 188 pages. Philadelphia, PA: American College of Physicians-American Society of Internal Medicine, 1998. $40.00.

In the preface, editors Raoof and Khan state that their manual is designed to be a quick reference source for interns or other practitioners interested in mechanical ventilation. This soft-cover book is in an outline format and divided into 3 sections.

Part I, "Why Ventilate?", briefly outlines the difference between ventilatory and oxygenation failure, the pathophysiologic indications, and special clinical conditions where ventilatory support is required. The objectives of mechanical ventilation are defined according to 1992 American Association for Respiratory Care and 1993 American College of Chest Physicians Mechanical Ventilator Consensus conferences.[1,2]

Part II, "How To Ventilate," discusses differences between positive and negative pressure ventilators, intensive care unit versus transport ventilators, and volume-targeted versus pressure-targeted modes of ventilation. In the chapter, "Basics of Initial Ventilator Set-Up," the authors review a selection of appropriate tidal volumes, oxygen concentrations, ventilator rates, and inspiratory flow. The effects of different inspiratory flows on inspiratory time and the problems associated with inappropriate flow are addressed. An illustration of insufficient inspiratory flow in the spontaneously breathing patient is given; the authors point out that this can be detected by observing a downward deflection of the ventilator airway manometer needle. Unfortunately, the authors do not complete the lesson by utilizing waveform graphics to document this same phenomenon. An example is provided (Appendix 2, Figure 56); however, the diagram's quality is marginal, and it fails to show the "scalloping out" of the pressure wave that is pathognomonic of this event.

Newer modes and techniques of mechanical ventilation are each described in this section. They include airway pressure release ventilation; proportional-assist ventilation and permissive hypercapnia; noninvasive positive pressure ventilation in acute respiratory failure; and the monitoring during, weaning from, and withdrawal of ventilatory support.

Complications associated with mechanical ventilation are outlined in Part III. The problems include volutrauma, hemodynamic compromise, nosocomial pneumonia, and oxygen toxicity. A chapter is included to deal with special problems encountered in specific clinical conditions. An algorithm is presented to aid trouble-shooting during mechanical ventilation, which may benefit the inexperienced practitioner. A section on nutritional support addresses the problem of malnutrition in the ventilated patient. The authors describe causes of malnutrition, its clinical importance, and how to assess the patient's nutritional status. A reference to the "resisting" energy expenditure should be corrected to read "resting" energy expenditure.

Part IV, the Appendix, is a valuable section of the manual, containing several sections that the respiratory care practitioner preparing for boards may find useful. These include guidelines on the parameters used to assess the need for and initial institution of ventilatory support, a section of formulae commonly used during mechanical ventilation, several compliance measurement exercises, and a 38-question quiz. Also included are 5 case studies covering several clinical conditions—including asthma, chronic obstructive pulmonary disease, acute respiratory distress syndrome, pulmonary edema, and a chapter of diagrammatic representations (which also offers an analysis of waveforms). Several figures in this section are poor examples (including Figure 56, mentioned earlier, and Figure 50, in which the inspiratory pressure versus time baseline changes without explanation). The waveform section illustrations appear to be computer-generated and do not meet the standards of quality that are found in the Graphics Corner of RESPIRATORY CARE. Though they do convey the basic points, actual ventilator-generated examples would better serve the authors and readers.

This final section also includes a glossary of terms and definitions that coincide with the 1993 ventilator consensus terminology and classification. The manual's chapters are not referenced. However, the annotated bibliography lists major contributors to the mechanical ventilator literature. References are current to 1997. The manual's outline form may not be as enjoyable to read as other books that use a narrative. A condensed or a pocket-size version may be better appreciated. Despite a few typographical errors and less than ideal illustrations, this manual does contain much of the essential information needed for care of the ventilated patient. Its use of graphs and algorithms helps convey concepts in simple understandable terms. The **Mechanical Ventilation Manual** does fill the requirements of residents or students of respiratory

care looking for basic reference material that is quick and easy to interpret.

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Design of Pulse Oximeters, JG Webster, Editor. (Medical Science Series) Hardcover, illustrated, 244 pages. Bristol UK and Philadelphia PA: Institute of Physics Publishing; 1997. $90.00.

This book emphasizes the design of pulse oximeters and is one of the Medical Science Series of the International Federation for Medical and Biological Engineering and the International Organization for Medical Physics. It includes the hardware and software required to fabricate the pulse oximetry as well as the equations, methods, and software required for effective functioning. The book is written for biomedical engineers, medical physicists, and health care providers.

The book consists of 244 pages divided into 13 chapters. A glossary is included at the end of the book. Each chapter includes relevant, but not exhaustive, references. Objectives are included with each chapter. Curiously, these are found at the end of the chapter rather than the usual placement at the beginning of the chapter. The editor and all of the chapter contributors are from the Department of Electrical and Computer Engineering at the University of Wisconsin.

The first 2 chapters of the book cover normal oxygen transport and the motivation for pulse oximetry. I suspect that many clinicians will find these chapters too superficial to be useful. Noticeably absent from this chapter is a discussion of mixed venous oxygen. I also expected some discussion of carboxyhemoglobinemia and methemoglobinemia, given that both of these hemoglobinopathies can affect pulse oximetry.

Chapter 3 provides an overview of blood oxygen measurement. The discussion of the Clark electrode is too superficial to be useful. Missing is discussion of how the Clark electrode is incorporated into the blood gas analyzer and issues such as calibration and quality control. The discussion of spectrophotometers and carbon monoxide-oximeters, however, is good. Mention of indwelling blood gas probes is nearly absent, without discussion of optodes or mixed venous oxygen saturation catheters. There is no analysis of point-of-care testing.

Light absorbance in pulse oximetry is based on Beer's law. Beer's law is used to determine the oxygen saturation of hemoglobin solutions, but does not apply for whole blood because of the scattering effects. Nonetheless, Beer's law provides a theoretical model to describe the principles of pulse oximetry. Chapter 4 provides a superb discussion of Beer's law as it applies to the measurement of oxygen saturation.

Chapters 5, 6, and 7 cover light-emitting diodes, photodetectors, and probes. Pulse oximetry requires a light source that is powerful enough to penetrate tissue, is small enough to fit into a probe, and has a narrow emission spectrum. These requirements are met by light-emitting diodes (Chapter 5). As discussed in Chapter 6, the amount of light that passes through tissue from the light-emitted diode is measured by a photodetector. The photodetector produces a current that is proportional to the intensity of the incident light. Issues related to light-emitting diodes and photosensors are discussed in extensive detail. Although this may be very important information for engineers responsible for designing pulse oximeters, it has limited usefulness for clinicians. The chapter on probes (Chapter 6) is more clinically useful. The probe packages the light-emitting diodes and photosensor so it can be attached to the patient. Clinicians must regularly choose an appropriate pulse oximetry probe and troubleshoot issues related to errors due to probes and their placement.

Chapter 8 covers electronic control of pulse oximetry and Chapter 9 covers signal processing algorithms. These chapters are very technical and thus might be of limited usefulness to clinicians.

Chapter 10 analyzes issues related to calibration of pulse oximeters. Clinicians will find this information useful. The section of the chapter on simulators to check the functionality of pulse oximeters is particularly good. Similarly, the chapter on accuracy and errors of pulse oximeters (Chapter 11) has clinical relevance. However, there is an important error in this chapter related to inaccuracies due to methemoglobin. It is stated that pulse oximeters overestimate the oxygen saturation in the presence of methemoglobin. This is only correct if the true oxygen saturation is less than 85%. Because methemoglobin causes a pulse oximetry saturation of 85%, it will produce an inaccurately low oxygen saturation reading if the true oxygen saturation is greater than 85% and it will produce an inaccurately high oxygen saturation if the true oxygen saturation is less than 85%.

Chapter 12 describes issues related to the user interface for pulse oximeters. Clinicians may find interesting the discussions of graphical displays, numerical displays, and alarm controls. The final chapter of the book (Chapter 13) describes clinical applications of pulse oximetry. There are interesting discussions of pulse oximetry applications in monitoring tissue blood supply and organ viability (such as dental pulp blood supply), use of pulse oximetry during ground and air transport, and fetal monitoring during childbirth. Noticeably absent from this chapter is a discussion of the use of pulse oximetry in the intensive care unit.. This chapter, like many parts of the book, reflects the fact that the authors are engineers and not clinicians.

The major strength of this book is its detailed coverage of the technical aspects of pulse oximetry. In that respect, the book has achieved its primary intent. However, this is at the expense of clinical relevance in many places throughout the book. Because of its strong engineering approach, mathematical relationships are presented throughout the text that clinicians might find intimidating. I would recommend this book for anyone who wishes to learn more about the technical aspects of pulse oximetry—ie, what happens inside the box between the probe and the display. Due to its relatively steep cost ($90) and limited clinical usefulness, this is not a book that will find its way onto the shelves of many respiratory therapists and critical care physicians.

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Human Immunodeficiency Virus and the Lung, Mark J Rosen MD and James M Beck MD, Editors. (Lung Biology in Health and Disease, Volume 119, Claude Lenfant, Executive Editor) Hardcover, illustrated, 584 pages. New York: Marcel Dekker Inc; 1998. $195.00.

One of the most impressive features of the acquired immunodeficiency syndrome (AIDS) epidemic is the rapidity at which knowledge on all aspects of human immunodeficiency virus (HIV) infection has evolved, including knowledge about the virus itself, the pathophysiology and natural history of HIV infection, and the syndromes associated with HIV-induced immunosuppression. Furthermore, this knowledge seems to be in a status of continuous change. These changes occur on many fronts, ranging from the epidemiology of HIV-infection and AIDS to the therapeutic approaches available for AIDS-related complications. Such a rapid pace of change makes it easy for the non-HIV-specialist to feel confused and bewildered when approaching the field of HIV disease. **Human Immunodeficiency Virus and the Lung**, co-authored by some of the most important authorities in the field, represents a major effort to summarize in a concise, yet complete fashion the current knowledge on HIV infection and its pulmonary complications and sequelae.

The book is divided into 6 different sections. Part One provides an overview of HIV infection, which, in addition to epidemiology, includes a chapter on HIV and injection drug users and a chapter dedicated to intensive care for patients with HIV infection. I found these chapters particularly important. While the initial spread of the AIDS epidemic occurred mostly within the gay community, the HIV is currently spreading at a faster rate among intravenous drug users and members of nongay minority groups. The chapter on HIV and injection drug users addresses both the specific complications of HIV in this particular group as well as the challenges confronting prevention and infection control strategies among intravenous drug users. The chapter dedicated to intensive care for patients with HIV infections includes some important ethical and moral considerations, such as the difficult situation raised when gay patients prefer their lovers or partners, rather than their biological family members, to act as surrogates.

Part Two discusses aspects of the cell and molecular biology of HIV infection that are relevant to the lungs. The section is quite appropriately divided into chapters dedicated to HIV and infection of lung cells, HIV and lung lymphocyte function, and HIV and alveolar macrophage function.

Parts Three, Four, and Five are dedicated to clinical aspects of HIV and the lungs. Part Three, "Diagnosis of Pulmonary Disorders," includes excellent chapters on bronchoscopy and on noninvasive diagnostic tests. The chapter on bronchoscopy includes useful diagnostic algorithms to approach focal and diffuse pulmonary disease, while the chapter on noninvasive tests includes a useful table on the radiographic patterns of abnormalities in HIV infection. The remaining clinical sections (Four and Five) include descriptions of infectious and noninfectious complications of HIV. These chapters provide excellent clinical descriptions of the different pulmonary syndromes associated with HIV, although it should be noted that the treatment (and prevention) of many of these entities is rapidly evolving. The chapter on *Pneumocystis carinii* pneumonia summarizes the experience at San Francisco General Hospital in a very useful algorithm for the diagnosis of this condition.

This book will be of interest for all health care providers involved in the treatment of HIV-infected patients. The target audience includes pulmonary and critical care physicians, respiratory therapists, and intensive care nurses. Respiratory therapists and nurses may find particularly interesting the chapters related to invasive and noninvasive diagnostic procedures, as well as the clinical sections of the book.

The most difficult problem faced by any book on HIV disease is that the rapidity at which the field evolves makes it extremely difficult for the authors to provide truly up-to-date information by the time the book is printed. The editors recognize this problem in the preface and state their efforts to "include the most comprehensive and current information available, emphasizing that which is unlikely to change by the time the book is published." Perhaps for this reason the most current Centers for Disease Control case definition of AIDS is not included (although it is referenced). Unfortunately, some important new findings failed to make their way into the pages of the book, particularly the role of chemokine receptors, such as CXCR5, as co-receptors for HIV. Also, I would have liked to see more discussion on the impact of highly active antiretroviral therapy (HAART) on the evolution of HIV-related pulmonary complications. However, overall the authors meet their stated goal of "providing a comprehensive compendium of [the pulmonary complications of HIV infection] and synthesize disparate information into an accessible format."

In general, the book is well written and easy to read. The index is well organized and facilitates the rapid location of specific contents. Numerous figures, photographs, and tables support and complement the text. The list of references is extensive (over 1,500), but few cover papers published after 1996. Unfortunately, there are an unusually high number of typographical errors, and in at least one occasion a missing word obscures the meaning of a sentence (Page 73).

In summary, in addition to providing an excellent review of both the infectious and noninfectious pulmonary complications of HIV, the book also addresses general aspects of the epidemiology and pathogenesis of HIV infection, aspects of cell and molecular biology of particular relevance to the lungs, and specific issues related to the care of the HIV patient with pulmonary disease or to the HIV patient in the intensive care unit. Health care providers involved in the care of HIV-infected patients will find particularly useful the chapters dedicated to the different pulmonary complications of HIV disease.

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New Products & Services

System Software for Exercise System. Spacelabs Burdick Inc announces the release of version 2.1 software for the Quest® Exercise Stress System. According to a company press release, the updated version includes several new features: graphical and tabular trends (both on display and printed components of the in-stage and final reports); test trended heart rate, blood pressure, and ST level and slope measurements during the exercise stress test; the system can now be interfaced with three different commercially available NIBP to take programmed BP mesurements and incorporate them into final reports; "freeze screen" function allows user to print and save 10-second, 12-lead printouts; and a Digital Baseline Stabilization Filter eliminates baseline wander artifact. For more information from Spacelabs Burdick, circle number 191 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Bronchodilator. Sepracor Inc has received final approval from the U.S. Food and Drug Administration (FDA) to market Xopenex™(levalbuterol HCl). According to the company, the inhalation solution is available in two dosage strengths for use with a nebulizer for the treatment and prevention of bronchospasm. Sepracor says the new drug is the therapeutically active (R)-isomer of racemic albuterol. A company spokesperson describes racemic albuterol as an equal mixture of (R) and (S) isomers and explains that although the drug is the world's leading brochodilator for asthma, they found that removing the (S) isomers increased effectiveness of the (R) isomer. For more information from Sepracor, circle number 192 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Vital Signs Monitor. BCI International has introduced its new vital signs monitor, the Advisor™. According to the company, the new monitor offers all of the basic parameters for vital signs monitoring, with ECG (3/5 lead), NIBP, S_{pO_2}, respiration, invasive pressures, and temperatures. BCI says a recorder and battery option are available and that a PC card allows for field upgrades. For more information from BCI International, circle number 193 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Nasal Wash System. Respironics has recently made available its RinoFlow™ Nasal Wash and Sinus System for sinus and allergy sufferers. A company press release says the device provides a gentle way to help relieve the pain and pressure associated with congested nasal or sinus passages. Respironics describes the RinoFlow as portable and easy to use, consisting of a small compressor unit the size of a tissue box (weighing about 5 lbs) and a hand-held misting chamber and tube that connect to the compressor. For more information from Respironics, circle number 194 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Page ___ of ___

Form Approved: OMB No. 0910-0291 Expires: 4/30/96
See OMB statement on reverse

FDA Use Only (Resp Care)

Triage unit sequence #

A. Patient information

1. Patient identifier
In confidence

2. Age at time of event:
or
Date of birth:

3. Sex
☐ female
☐ male

4. Weight
___ lbs
or
___ kgs

B. Adverse event or product problem

1. ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2. Outcomes attributed to adverse event (check all that apply)
☐ death ___ (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other: ___

3. Date of event (mo/day/yr)

4. Date of this report (mo/day/yr)

5. Describe event or problem

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)
#1
#2

2. Dose, frequency & route used
#1
#2

3. Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4. Diagnosis for use (indication)
#1
#2

5. Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)
#1
#2

7. Exp. date (if known)
#1
#2

8. Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. NDC # (for product problems only)
– –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1. Brand name

2. Type of device

3. Manufacturer name & address

4. Operator of device
☐ health professional
☐ lay user/patient
☐ other: ___

5. Expiration date (mo/day/yr)

6.
model # ___
catalog # ___
serial # ___
lot # ___
other # ___

7. If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9. Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ___ (mo/day/yr)

10. Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. Name & address

phone #

2. Health professional?
☐ yes ☐ no

3. Occupation

4. Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

FDA Form 3500 1/96

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burdan for this collection of information has bean estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data neadad, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

DHHS Reports Clearance Office
Paperwork Reduction Project (0910-0291)
Hubert H. Humphrey Building, Room 531-H
200 Independence Avenue, S.W.
Washington, DC 20201

"An agency may not conduct or sponsor, and a parson is not raquired to respond to, a collaction of information unless it diaplays a currantly valid OMB control number."

Please do NOT return this form to either of these addresses.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service • Food and Drug Administration

FDA Form 3500-back

Please Use Address Provided Below – Just Fold In Thirds, Tape and Mail

Department of Health and Human Services
Public Health Service
Food and Drug Administration
Rockville, MD 20857

Official Business
Penalty for Private Use $300

NO POSTAGE NECESSARY IF MAILED IN THE UNITED STATES OR APO/FPO

BUSINESS REPLY MAIL
FIRST CLASS MAIL PERMIT NO. 946 ROCKVILLE, MD

POSTAGE WILL BE PAID BY FOOD AND DRUG ADMINISTRATION

MEDWATCH
The FDA Medical Products Reporting Program
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20852-9787

American Association for Respiratory Care

MEMBERSHIP APPLICATION

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

- ☐ Active
- Associate
  - ☐ Foreign
  - ☐ Physician
  - ☐ Industrial
- ☐ Special
- ☐ Student

Last Name _____

First Name _____

Social Security No. _____

Home Address _____

City _____

State _____ Zip __ __ __ __ __ — __ __ __ __

Phone No. (_____) _____

Primary Job Responsibility (check one only)

- ☐ Technical Director
- ☐ Assistant Technical Director
- ☐ Pulmonary Function Specialist
- ☐ Instructor/Educator
- ☐ Supervisor
- ☐ Staff Therapist
- ☐ Staff Technician
- ☐ Rehabilitation/Home Care
- ☐ Medical Director
- ☐ Sales
- ☐ Student
- ☐ Other, specify _____

Type of Business

- ☐ Hospital
- ☐ Skilled Nursing Facility
- ☐ DME/HME
- ☐ Home Health Agency
- ☐ Educational Institution
- ☐ Manufacturer or supplier
- ☐ Other, specify _____

Date of Birth (optional) _____ Sex (optional) _____

U.S. Citizen? ____ Yes ____ No

Have you ever been a member of the AARC? _____

If so, when? From _____ to _____

Preferred mailing address: ☐ Home ☐ Business

For office use only

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Medical Director/Medical Sponsor _____

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

School/RC Program _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Length of program

- ☐ 1 year
- ☐ 2 years
- ☐ 4 years
- ☐ Other, specify _____

Expected Date of Graduation *(REQUIRED INFORMATION)*

Month _____ Year _____

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • [972] 243-2272 • Fax [972] 484-2720

American Association for Respiratory Care MEMBERSHIP APPLICATION

Demographic Questions

We request thot you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRT
- ☐ Physicion
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
- ☐ CPFT
- ☐ RPFT
- ☐ Perinatal/Pediatric

Salary

- ☐ Less than $10,000
- ☐ $10,001-$20,000
- ☐ $20,001-$30,000
- ☐ $30,001-$40,000
- ☐ $40,000 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respirotory Core and hove enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws ond professional code of ethics. I outharize investigation of all statements contained herein and understand that misrepresentations or omissions of facts colled for is couse for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and *AARC Times* magazine includes on allocation of $11.50 from my dues for each of these publicotions.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association labbying activities. The AARC estimotes that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature _____

Date _____

Membership Fees

Payment must accompany your applicotion to the AARC. Fees are for 12 months. (NOTE: Renewal fees ore $75.00 Active, Associate-Industrial or Associate-Physician, or Special status; $90.00 for Associate-Foreign status; and $45.00 for Student status).

☐ Active	$ 87.50
☐ Associate (Industrial or Physician)	$ 87.50
☐ Associote (Foreign)	$102.50
☐ Speciol	$ 87.50
☐ Student	$ 45.00
TOTAL	$_____

Specialty Sections

Established to recognize the specialty areas of respiratory care, these sections publish a bi-monthly newsletter thot focuses on issues of specific concern to thot specialty. The sections olso design the specialty programming at the national AARC meetings.

☐ Adult Acute Care Section	$15.00
☐ Educotion Section	$20.00
☐ Perinatol-Pediotric Section	$15.00
☐ Diagnostics Section	$15.00
☐ Continuing Care-Rehabilitation Section	$15.00
☐ Management Section	$20.00
☐ Transport Section	$15.00
☐ Home Care Section	$15.00
☐ Subacute Core Section	$15.00
TOTAL	$_____

GRAND TOTAL = Membership Fee plus optional sections $_____

- ☐ Total Amount Enclosed/Charged $_____
- ☐ Please charge my dues (see below)

To charge your dues, complete the following:

- ☐ MasterCord
- ☐ Visa

Card Number

— — — — — — — — — — — — — — — —

Card Expires _____/_____

Signature _____

Mail application and appropriate fees to:

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • [972] 243-2272 • Fax [972] 484-2720

call for abstracts

1999 RESPIRATORY CARE OPEN FORUM

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in Las Vegas, Nevada, December 13–16, 1999. Accepted abstracts will be published in the November 1999 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract is the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions*. Give specific information. Do not write general statements, such as "Results will be presented" or "Significance will be discussed."

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocol evaluation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

FORMAT AND TYPING INSTRUCTIONS

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. *Abstracts should be 400 words or less and may have 1 clear, concise table or figure.* A font like Helvetica or Geneva makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in a single paragraph in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. *Data may be submitted in table form, or a simple figure may be included provided it fits within the space allotted. No figure, illustration, or table is to be attached to the abstract form.* Provide all author information requested. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (972) 406-4667.

Early Deadline Allowing Revision. Authors may choose to submit abstracts early. Abstracts postmarked by April 2, 1999 will be reviewed and the authors notified *by letter only* to be mailed by May 7, 1999. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the Final Deadline (June 11, 1999).

Final Deadline. The mandatory Final Deadline is June 11, 1999 (postmark). Authors will be notified of acceptance or rejection *by letter only*. These letters will be mailed by August 25, 1999.

Mailing Instructions. Mail (do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

1999 RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593

submit your OPEN FORUM abstract electronically
visit www.rcjournal.com

1999 RESPIRATORY CARE OPEN FORUM Abstract Form

18.8 cm or 7.4"

13.9 cm or 5.5"

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with all authors' names, including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, leave a 'ragged' right margin).
4. **Do not use type size less than 10 points.** (Do not exceed 400 words.)
5. All text and the table, or figure, must fit into the rectangle shown. (Use only 1 clear, concise table or figure.)
6. Submit 2 clean copies. This form may be photocopied if multiple abstracts are to be submitted.

Mail original & 1 photocopy (along with postage-paid postcard) to

1999 RESPIRATORY CARE OPEN FORUM
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Corresponding Author If Different from Presenter

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RESPIRATORY CARE

Manuscript Preparation Guide

General Information

RESPIRATORY CARE welcomes original manuscripts related to the science and technology of respiratory care and prepared according to these Instructions and the *Uniform Requirements for Manuscripts Submitted to Biomedical Journals* [Respir Care 1997; 42(6):623-634]. Manuscripts are blinded and reviewed by professionals who are experts in their fields. Authors are responsible for all aspects of the manuscript and receive galleys to proofread before publication. Each accepted manuscript is copyedited so that its message is clear and it conforms to the Journal's style. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission.

Editorial consultation is available at any stage of planning or writing. On request, specific guidance is provided for all publication categories. To receive these Instructions and related materials, write to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104, call (206) 223-0558, or fax (206) 223-0563.

Publication Categories & Structure

Research Article: A report of an original investigation (a study). It includes a Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Evaluation of Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification. It has a Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions. Comparative cost data should be included wherever possible.

Case Report: A report of a clinical case that is uncommon, or was managed in a new way, or is exceptionally instructive. All authors must be associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript. Its components are Title Page, Abstract, Introduction, Case Summary, Discussion, References, Tables, Figures, and Figure Captions.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References. Tables, Appendices, and Figures and Captions may be included.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles.

Update: A report of subsequent developments in a topic that has been critically reviewed in this Journal or elsewhere.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Title Page, Text, References, Tables, and Illustrations may be included.

Special Article: A pertinent paper not fitting one of the foregoing categories may be acceptable as a Special Article. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A signed communication, marked "For publication," about prior publications in this Journal or about other pertinent topics. Tables and illustrations may be included.

Blood Gas Corner: A brief, instructive case report involving blood gas values—with Questions, Answers, and Discussion.

Drug Capsule: A mini-review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, and pharmacotherapy.

Graphics Corner: A brief case report incorporating waveforms for monitoring or diagnosis—with Questions, Answers, and Discussion.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Cardiorespiratory Interactions. A case report demonstrating the interaction between the cardiovascular and respiratory systems. It should be a patient-care scenario; however, the case—the central theme—is the systems interaction. CRI is characterized by figures, equations, and a glossary. See the March 1996 Issue of RESPIRATORY CARE for more detail.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs; may involve imaging techniques other than conventional chest radiography.

Review of Book, Film, Tape, or Software: A balanced, critical review of a recent release.

Preparing the Manuscript

Print on one side of white bond paper, 8.5 in. x 11 in. (216 x 279 mm) with margins of at least 1 in. (25 mm) on all sides of the page. Use ***double-spacing*** throughout the ***entire*** manuscript. Use a standard font (eg, Times, Helvetica, or Courier) at least 10 points in size, and

do not use italics except for special emphasis. Number all pages in upper-right corners. Indent paragraphs 5 spaces. Do ***not justify***. **Do *not* put authors' names, institutional affiliations or allusions to institutional affiliations in the text, or other identification anywhere except on the title page.** Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: Title Page, Abstract, Text, Product Sources List, Acknowledgments, References, each Table, and each Appendix. Use standard English in the first person and active voice.

Center main section headings on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin subheadings at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis).

References. Cite only published works as references. Manuscripts accepted but not yet published may be cited as references: designate the accepting journal, followed by (in press), and provide 3 copies of the in-press article for reviewer inspection. Cite references in the text with superscript numerals. Assign numbers in the order that references are first cited. On the reference page, list the cited works in numerical order. Follow the Journal's style for references. Abbreviate journal names as in *Index Medicus*. List all authors.

Article in a journal carrying pagination throughout volume:

> Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

> Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

> American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

> Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986; 89(3 Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

> Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

> Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

> Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

> Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

> Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

> DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990. p. 76-85.

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> American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

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> Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott; 1977:26-42.

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Calendar of Events

AARC & AFFILIATES

April 27—*Teleconference*
After viewing a tape of the second installment of the AARC's 1999 "Professor's Rounds" series, "Asthma: Managing the Disease," participate in a live telephone question-and-answer session (11:30–12 noon CDT) and receive one CRCE credit hour.
Contact: The AARC at (972) 243-2272.

April 30—*Edinboro, Pennsylvania*
The Northwest District of the PSRC will host its 18th Annual Education Seminar and Equipment Exhibition at the Ramada Inn Edinboro. Featured speaker is Thomas Petty, MD, presenting "Excerpts from the Evolution of Pulmonary Medicine: Looking Ahead to the Solution of COPD."
Contact: Steve Easly at (814) 866-3555.

April 30–May 2—*Philadelphia, Pennsylvania*
The AARC will be conducting the Patient Assessment Course for respiratory therapists at the Westin Suites Philadelphia Airport. Each attendee will receive a pocket guide to physical assessment, and 16 hours of CRCE credit are available as well as a certificate of course completion. Preregistration is required.
Contact: The AARC at (972) 243-2272.

May 2–4—*Bellevue, Washington*
The Respiratory Care Society of Washington announces the 26th Annual Pacific Northwest Regional Respiratory Care Conference and exhibit at the Meydenbauer Center. Speakers include forensics expert Dr. Henry Lee, AARC neonatal/pediatrics section chair Katie Sabato, and AARC spokesperson Cheryl West. CRCE credit available.
Contact: Ellen Perry at (425) 899-3361 or e-mail **ezperry@earthlink.net**.

May 4–5—*Hartford, Connecticut*
The CSRC present their Symposium '99 — "Confidence in Tomorrow" at the Foxwoods Resort and Casino. The keynote speaker is Richard Blumenthal, Connecticut Attorney General; and featured speaker is Dr. Henry Lee, renowned forensic scientist.
Contact: John Evenwell at (203) 688-2201 for more information.

May 19–21—*El Paso, Texas*
The Southwest Region of TSRC is sponsoring its 28th Annual Seminar at the El Paso Community College, Transmountain Campus. Outstanding speakers and state-of-the-art equipment will be featured, and up to 12 CE hours are available.
Contact: Bertha Butler at (915) 577-6563.

May 19–21—*Virginia Beach, Virginia*
The VSRC announces its 22nd annual spring seminar, "Symposium by the Sea," at the Ramada Tower at the Oceanfront. Sam Giordano is the keynote speaker.
Contact: Dolly Saunders at (757) 468-0702.

May 20–21—*Rockport, Maine*
The MSRC presents "The Maine Event" at the Samoset Resort. Topics include ER trauma, neonatal waveforms, pulmonary rehab, the tobacco settlement, and HMOs/managed care.
Contact: Bobbi Shirley at (207) 797-0793 for more information.

May 28—*San Antonio, Texas*
The University of Texas Health Science Center at San Antonio, in conjunction with the TSRC (Alamo District) and Wilford Hall Medical Center, announce the 4th Annual Riverwalk Respiratory Care Symposium at the Sheraton Four Points Hotel Riverwalk North. Topics include disease management, respiratory care in the year 2005, neonatal update, managing cystic fibrosis, and critical care transport. Six CRCE credits provided.
Contact: UTHSCSA, Department of Respiratory Care at (210) 567-8850.

June 16–18—*Oakbrook, Illinois*
The 1999 ISRC annual convention and exhibition will be held at Drury Lane Theatre and Conference Center in the Chicagoland area. Topics will include management and subacute, with opportunities under PPS, maximizing career potential and survival techniques for respiratory care. Breakout sessions include mechanical ventilation, blood gas regulation update, and CO-oximetry.
Contact: Mike Lawrence at (618) 234-2120, ext. 1287, or e-mail **mlawrence@lclsnet.lcls.lib.il.us**.

Other Meetings

May 6–8—*Las Vegas, Nevada*
The American Lung Association of Nevada is sponsoring the 14th annual Respiratory Health Conference at the Tropicana Resort and Casino.
Contact: Call (702) 431-6333 for more information.

June 12-16, 1999—*International Society for Aerosols in Medicine*
12th International Congress at the Austria Center in Vienna, Austria. Topics include aerosol drug delivery, aerosol deposition and clearance, cellular and molecular interactions, environmental aerosols, standardization, aerosol diagnostics, and aerosol therapy.
Contact: Vienna Academy of Postgraduate Medical Education and Research. Alser Strasse 4, A-1090 Vienna, Austria. Phone (+43/1) 405 13 83-22, fax (+43/1) 405 13 83-23, e-mail medacad@via.at.

August 19–20—*Cleveland, Ohio*
The Cleveland Clinic Foundation is sponsoring a continuing education program titled "Respiratory Therapy," which has been approved for Category 1 accreditation. It will be held at the Omni International Hotel.
Contact: For more information, call Laurie Martel at (216) 444-5696 or (800) 862-8173.

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org
- —Current job listings
- —American Respiratory Care Foundation fellowships, grants, & awards
- —Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

RESPIRATORY CARE online
http://www.rcjournal.com
- —1997 Subject and Author Indexes
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Asthma Management Model System
http://www.nhlbi.nih.gov

The National Board for Respiratory Care—1999 Examination Dates and Fees

Examination	Examination Date	Examination Fee
CRT Examination	July 10, 1999 Application Deadline: May 1, 1999	$120 (new applicant) 80 (reapplicant)
RRT Examination	December 4, 1999 Application Deadline: August 1, 1999	120 written only (new applicant) 80 written only (reapplicant) 130 CSE only (all applicants) 250 Both (new applicants) 210 Both (reapplicants)

For information about other services or fees, write to the National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156,or e-mail: nbrc-info@nbrc.org

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In this issue
1999 Call For Abstracts
See page 455

New Additions to AARC Web Site Make Communication Easy

The AARC's web site (www.aarc.org) make communication with the AARC Executive Office and among other AARC members much more direct and accessible. Recent additions to the site include:

Chat — AARC members can chat in real time. Organized chats on specific topics will be planned in the future.

Just Ask — Do you have a question about AARC policies or positions on issues? Do you need help in interpreting reimbursement and government policies? Do you want to know what the AARC is doing about legislative advocacy? You can post a question in this area for possible posting.

Hotline to the President — Do you want immediate action from the top? Click on the "red phone" hotline to President Dianne Kimball. An E-mail will be sent directly to her.

Help Line — Do you have a clinical or professional question you want answered? Post it on the help line and other AARC members will respond.

Specialty Section Mailing Lists — If you are a member of one of the nine specialty sections, you have instant networking capabilities through the electronic mailing lists of each group.

Patient Assessment Course for Respiratory Therapists

Due to overwhelming demand, the patient assessment course for respiratory therapists is being offered twice this year. The first test date has passed, and the remaining test will be conducted in Phoenix, Arizona from July 18-20 (immediately following Summer Forum). Space is at a premium and preregistration is required. Successful completion of the course will earn participants 16 hours of CRCE credit and a certificate of course completion. Each attendee will be given a pocket guide to physical assessment, to help them on the job. Following the last class, participants will take a 100-item test developed by the NBRC. Tests will be graded on-site for those wishing to obtain their scores immediately. For more information and to register, visit the AARC web site at www.aarc.org.

AARC, Affiliates Set Conference Schedules

The AARC and many of the affiliates have set their schedules for 1999 conferences and seminars. Foremost among AARC's offerings are its Summer Forum (July 16-18) and Annual International Respiratory Congress (Dec. 13-16). Check out the AARC's website at www.aarc.org for all meeting registration materials and a list of affiliate conferences.

Videoconference Program Set; Nursing CEUs Offered

A series of eight videoconferences are scheduled for 1999 through the AARC Professor's Rounds series, which are now approved for nursing CEUs as well as CRCE credit. Topics are: respiratory assessment, asthma management, ventilator management, disease management, pediatric emergencies, COPD, PEEP, and respiratory pharmacology.

CRCE Online Debuts

Now you can earn continuing education on the Internet from the AARC through its new CRCE Online website. After you pay for the number of continuing education units you wish to attempt (by submitting your credit card number on a secure server site), you are given access to the list of courses. Read the material, take the test, and then print out a certificate showing you passed. Your participation will also be noted on your CRCE record with the AARC. Log on to the AARC's website at www.aarc.org and look for CRCE Online.

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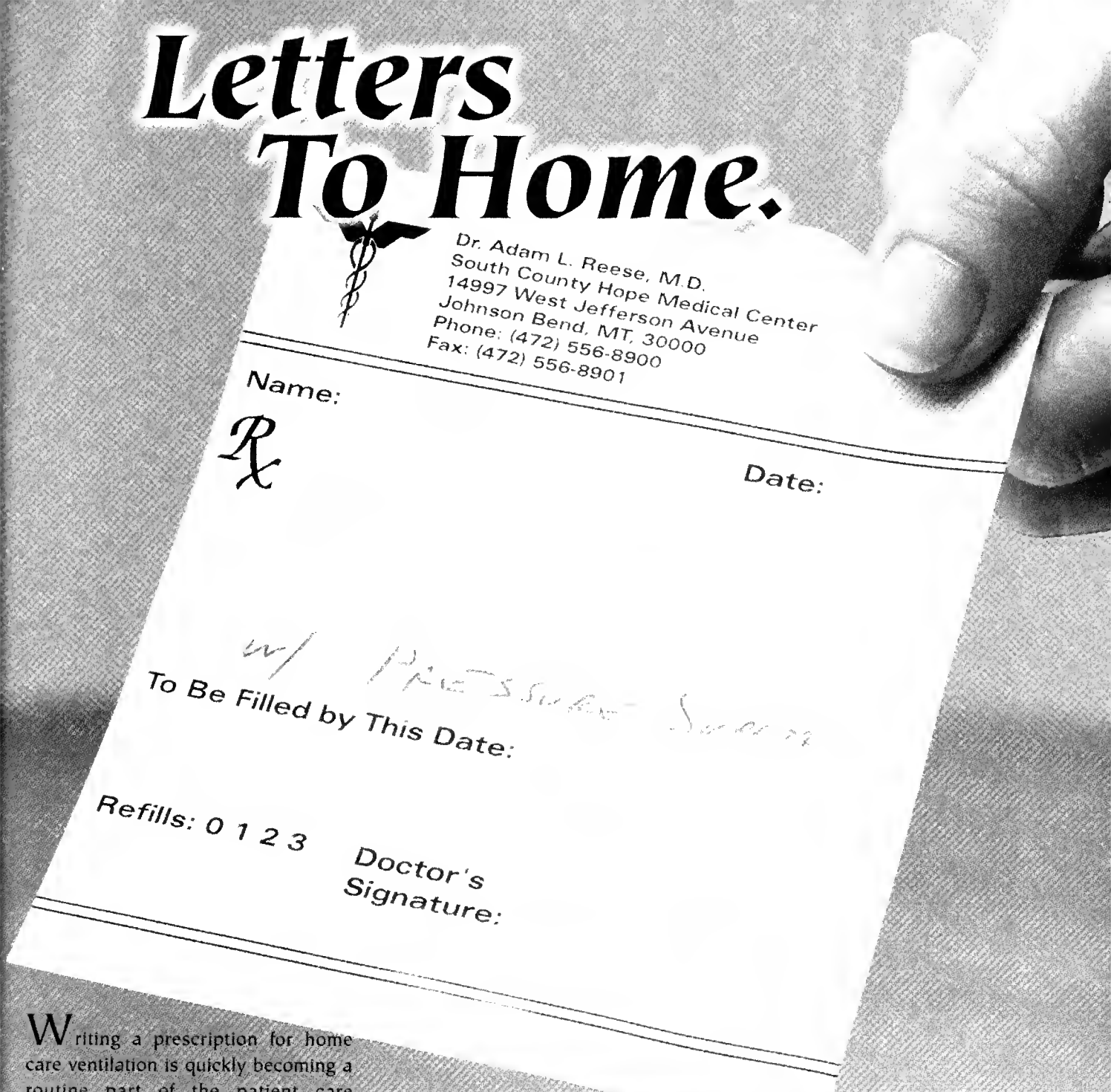

Writing a prescription for home care ventilation is quickly becoming a routine part of the patient care continuum. Unfortunately, equipment limitations have made caring for some patients more of a challenge than necessary.

Until Now.

Introducing the TBird Legacy, the *only* homecare ventilator delivering Pressure Support. Research has demonstrated Pressure Support's ability to reduce work of breathing.[1] Patients less fatigued by the ventilator can concentrate on getting well sooner, continuing the weaning process even at home.

Combine the value of Pressure Support with other features like flow triggering and integrated alarms and the result is a great homecare ventilator for your patients and their families. The Legacy's simplified patient interface even reduces the substantial burden placed on family members.

While numerous factors enter into your decision to send a patient home, the TBird Legacy's capabilities will help *–not hinder–* the decision. Write a prescription for TBird Legacy and give your patients hospital performance with all the comforts of home.

To learn more about effective homecare ventilation, contact Thermo Respiratory Alternate Care at (877) TRG-TRAC (874-8722).

BIRD®

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1100 Bird Center Drive, Palm Springs, CA 92262
Phone: (760) 778-7200 Fax: (760) 778-7274

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1. Branson, R.D., RRT & Davis, K., MD (1995). Work of breathing imposed by five ventilators used for long-term support; the effects of PEEP and simulated patient demand. Respiratory Care (Vol. 40, 12).

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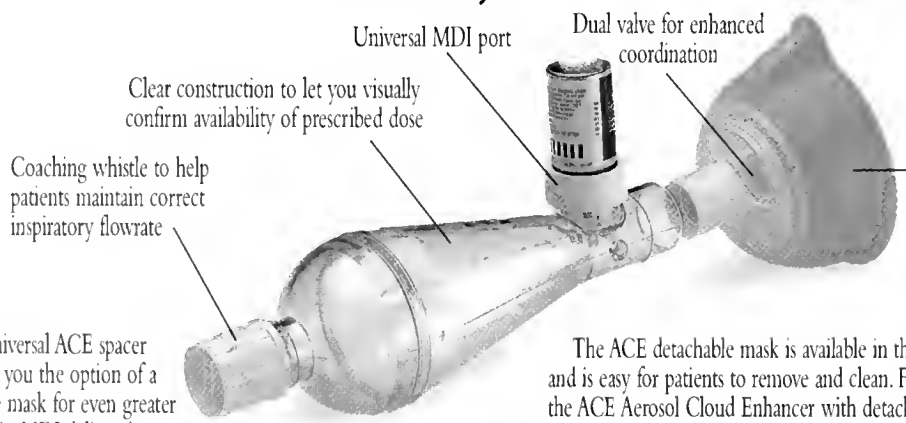
Now you need it.

Now you don't.

Introducing the ACE® Aerosol Cloud Enhancer with exclusive, *detachable* mask.



The universal ACE spacer now gives you the option of a detachable mask for even greater versatility in MDI delivery!

ACE eliminates the need to stock multiple MDI spacers. In addition to using ACE to deliver aerosolized medications through a mask, you can also use it in a vent circuit, in conjunction with an endotracheal airway, connected to a resuscitation bag, and with a mouthpiece for routine, oral therapy. No other spacer lets you deliver relief in more applications!

The ACE detachable mask is available in three sizes (sold separately), and is easy for patients to remove and clean. For more information on the ACE Aerosol Cloud Enhancer with detachable mask, or for a *free* catalog of DHD quality respiratory products, call toll-free today at **1•800•847•8000.**

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